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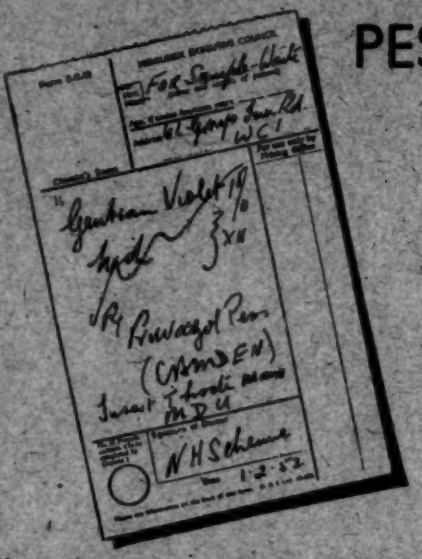

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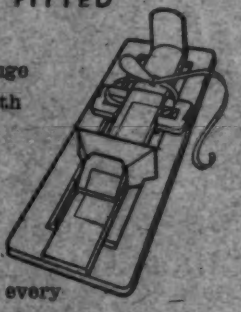

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BRITISH MEDICAL JOURNAL

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# CORONARY HEART DISEASE IN MEDICAL PRACTITIONERS

BY

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In a previous paper some aspects of the recent history of coronary disease were examined. The evidence given there supported the widely held view that there has been some true increase of coronary thrombosis and coronary heart disease during the present century, apart from the effect of the ageing of the population, and it was suggested also that this increase may not have been due to an increase of the underlying chronic atheroma (Morris, 1951). The present report turns to the contemporary situation, and gives some results of an inquiry on coronary heart disease carried out in various occupations.

There is no doubt that coronary heart disease is a major problem of the public health, and, however the certified causes of death are regarded, coronary heart disease must now be among the commonest. Little is known about its causes, and experience of other conditions suggests that examination of the epidemiology may well be rewarding. There are indications, also, from clinical experience and the figures of the Registrar-General, of social factors in coronary heart disease, in work and mode of life, and these clues must be followed up. If coronary heart disease is in fact commoner than it used to be, environmental *changes* with which the increase may be associated must be sought, however indirectly such changes may seem to operate.

An investigation has been conducted in several occupational groups; and the results of this study in one group, medical practitioners, are now being given because the information was obtained in a special way and because it is likely to be exceptionally reliable. There is some general feeling, too, that doctors may suffer more than others from coronary heart disease, and the present figures may give some idea, at least, of the "ceiling" of this disease in the population. This occupation, moreover, though of particular interest, is amongst the least studied.

## Material and Methods

The present study was made from the records of the Medical Sickness, Annuity and Life Assurance Society, Ltd. (after this called "the Society"), a mutual benefit organization which has several thousand doctors on "non-cancellable" sickness and accident policies (Heath, 1939). "Non-cancellable" indicates a form of insurance in which renewal is not subject to annual review by the Society, and continuance of the policy is thus independent of morbidity experienced while holding the policy. The Society's records of the later attacks of a disease, therefore, are as representative as its records of the first attacks. Most policies issued provide for payment of a weekly benefit from the outset of any sickness, or accident, causing total incapacity to carry out the member's occupation for seven days or longer. The inquiry was limited to the records of members holding these "immediate benefit" policies, since the records of other policies providing "deferred benefit" do not give full information concerning the early part of a period of incapacity. The Society's policies in general terminate at age 60 or at age 65 years, and the present inquiry is restricted to ages 35–64 years, inclusive. Males only were studied because of the small number of women in the Society in the age groups most important to this inquiry. Throughout the report, therefore, when "medical practitioners" or "doctors" are mentioned, reference is intended to male members of this Society, of the ages stated, holding the relevant insurance—and to no others.

The "population" being studied, although it is substantial and may include about a quarter of the country's doctors in the ages considered, is *selected*, because a full medical examination is given to all applicants. It is possible, therefore, for instance, that doctors who know they have diabetes or material hypertension may hesitate even to apply for insurance. The great majority of policy-holders, however, join in their late twenties and early thirties—that is, before coronary heart disease, or the conditions with which it may be associated, can usually be recognized. Such selection as this is likely to be in the "safe direction" for the present inquiry, for it will reduce the number of cases found, and not exaggerate it. There is no information on which to base discussion of the more subtle selective factors. Is it the more anxious personality who applies for a policy? Or the more careful? Or the man who is less careful but who knows himself to be so, and wishes to protect his dependants from the risks he cannot stop himself taking? Does an unhealthy family history prompt the taking out of insurance? There is even less basis on which to discuss the unconscious motivation involved. The mortality of members of this Society cannot be compared with that of all "physicians and surgeons, etc.," reported in the Registrar-General's occupational

mortality supplement for 1930–2 (Registrar-General, 1938), because the age structure of the Society's membership during those three years is unknown.

Payment of benefit is on the basis of medical certificates which involve at least two doctors—the patient and his medical attendant. Although a "specialist opinion" is not of course necessary, examination of a large proportion of the records of first illness from coronary heart disease showed that such confirmation of the diagnosis had, in fact, been recorded in three-quarters of the cases, often with copies of electrocardiographic findings, etc. As a routine precaution all the certificates for each illness were considered and the final diagnosis was used. The recording was carried out with a combination of serial numbers and names, which permitted the fullest use of the available information, and at the same time preserved the confidential nature of the records. All deaths of members are known to the Society, including coroners' cases, and a copy of the death certificate is obtained.

*Coronary heart disease* in the present inquiry includes the following disorders arising on atheroma of the coronary arteries, with thrombosis, obstruction, occlusion: the clinical syndromes usually called "*coronary thrombosis*" (*sudden deaths* and *myocardial infarction*); *angina pectoris* (or of effort); and occasional cases described as "*coronary insufficiency*," etc. Cases diagnosed as "angina pectoris," without a pathology being stated, were also included. "Ischaemic heart disease" is the more accurate term, but "coronary heart disease" is convenient and in too common use to be discarded. Throughout the paper reference is intended only to the above *clinical* manifestations of the disease, together with their complications and consequences, and not at all to the natural history of the disease in *pathological* terms. "First attack," for example, and "first clinical attack" are used interchangeably.

Apart from the main statements on the certificates, referring in the present instance to coronary heart disease, further information was often given—for example, on hypertension and other associated conditions. No particular attention, however, can here be paid to these. The present type of study, moreover, is not suitable for the consideration of such clinical matters as the site of infarction. The mortality analysis, similarly, deals only with coronary heart disease as the certified cause of death; often there was evidence of necropsy, but this aspect had also to be ignored.

In summary, therefore, information about the male members of the Society aged 35–64 years who hold immediate benefit non-cancellable sickness insurance, and are absent from work because of coronary heart disease, is available as follows: *Where the absence lasts less than a week:* Information only about *deaths*, from death certificates (and, often, other reports). *Where the absence lasts a week or longer:* Information on medical certificates about the *illness*, from the *first* day of the absence. Date of ending of *absence*, as well as its beginning. Information about *deaths* as above.

Figures will be presented for *morbidity* and *mortality*. The annual rates of occurrence of various manifestations of the disease at different ages will be worked out in the usual way, by reference to the total number of men concerned—for example, if over a given period there is an average of 12 cases a year (the "numerator") in men aged 50–54 years, and over the same period an average "population" of 1,500 doctors of that age with non-cancellable insurance (the "denominator"), the *average annual rate* is 8 per 1,000.

Since *comparison of rates* in two population groups can be misleading if one group includes more old people than the other, the rates in this paper which summarize the experience of men over a wide age range—for example, 40–59 years—will be *standardized* for age. The "standard" population that will be used here is the civilian population of England and Wales in mid-1949, the latest figures available at the time from the Registrar-General.

## PART I: THE DISEASE IN MEDICAL PRACTITIONERS

### Incidence

The "incidence" gives the number attacked by coronary heart disease for the first time, so far as is known, in 1,000 of these medical practitioners per year. The number of new cases is a basic figure to have in studying disease in any "population," whether it is this group of doctors or one doctor's practice. As used here, the "incidence" is the rate of all first manifestations of coronary heart disease, regardless of the form taken, that (*a*) were recognized and diagnosed among these medical practitioners, and (*b*) led to absence from work lasting a week or more, or to death. The attack was regarded as a first attack if there had been no previous claim for benefit for coronary heart disease.*

Column 1 of Table I shows the ages; column 2 the average number of practitioners holding the relevant insurance

TABLE I.—*Incidence of Coronary Heart Disease in Medical Practitioners, 1947–50. Men Aged 35–64 Inclusive. Average Annual Rates per Thousand*

| Ages Last Birthday (Years) (1) | Mean Population (2) | No. of First Attacks (3) | Incidence—First Attack Rate p.a. (4) |
|---|---|---|---|
| 35–39 | 1,155 | 2 | — |
| 40–44 | 1,367 | 12 | 2·2 |
| 45–49 | 1,549 | 21 | 3·4 |
| 50–54 | 1,006 | 28 | 7·0 |
| 55–59 | 696 | 33 | 11·9 |
| 60–64 | 407 | 27 | 16·6 |
| All ages 35–64 incl. | 6,180 | 123 | 5·0 |
| *Standardized rate* at ages | 40–59 incl. | | 5·5 |
| " " " " | 40–64 " | | 7·1 |

*Ages.*—All recording was based on year of birth. Ages are therefore in fact ages attained at the beginning of the calendar year.

*Mean Population.*—This was calculated from annual counts of the Society's membership, as on January 1 of the five years 1947–51. All men included held non-cancellable sickness and accident insurance, at the date of the count, of a type providing benefit from the beginning of periods of absence from work because of sickness which lasted seven days or more. The members of the Society are mostly resident in the United Kingdom, though there are a few elsewhere.

*First Attacks.*—These are men clinically attacked by coronary heart disease (as defined) for the first time between January 1, 1947, and December 31, 1950, who, at the time of this attack, held insurance of the type above. The figures are made up of (*a*) non-fatal first attacks causing absences from work for seven days or more, and (*b*) *all* fatal first attacks.

*Incidence.*—First attacks per 1,000 living, per annum.

*Standardized Rates.*—These are based on the civilian population of England and Wales in mid-1949.

*It is not possible to be absolutely certain that first claims for coronary heart disease always represented first absences for this condition, or that first absences always coincided with the first diagnosis, or first diagnoses with first clinical attacks. Diagnoses may be missed, and atypical and premonitory cardiac pain, for instance, may be overlooked. It is conceivable, too, that angina pectoris, even when diagnosed for the first time, and even among medical practitioners, did not always lead to as much as a week's sick leave. It is not possible, however, to check this kind of matter, and since it is unlikely that such reservations could be common enough to affect the age or other trends being investigated, they will be ignored. The further possibility that practitioners may have taken time from work, and not troubled to claim the benefit to which they were entitled, is also not likely to have modified the results. The Society's experience of claims made retrospectively is that such claims have rarely to be made for illnesses which have lasted any length of time. Eighty per cent. of observed first absences for angina pectoris, and 97% of observed first absences for "coronary thrombosis" (that was not rapidly fatal) lasted more than a month. The *average* first absences were, of course, much longer. In brief, it is considered that the chain of events, first diagnosis of coronary heart disease—sick absence for a week or longer—claim for benefit, is reasonably complete. The "sudden" deaths are in a different category, and are covered.

in each age group over the four years 1947–50; column 3 is the number of first attacks recorded in the four years; and column 4 gives the "incidence"—the annual rate of first attack per 1,000 in each age group. Column 4 shows a steep incline from the late thirties to 16.6 per 1,000 among men in their early sixties (Fig. 1).

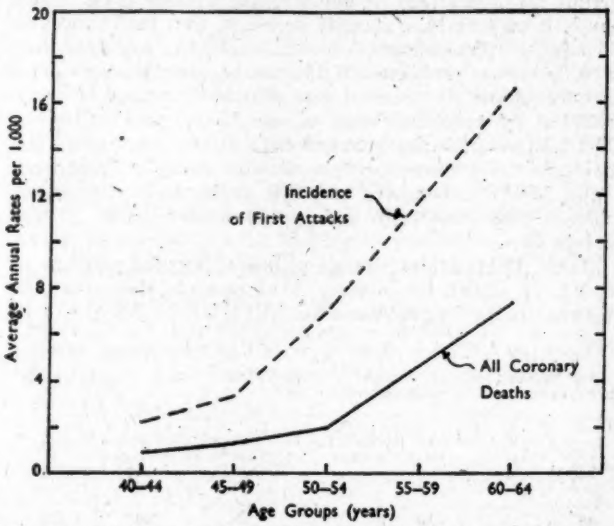


FIG. 1.—Incidence and mortality in coronary heart disease, 1947–50. Male medical practitioners aged 40–64 inclusive. (See notes to Tables I and II for information about the Society's membership, etc.)

## Mortality Rates

Coronary heart disease is the main "cause of death" in the Society after 35 years of age, about one-third of all mortality among members being ascribed to it, and this fraction does not vary with age. Under, say, 50 years this fraction will be influenced by the Society's policy of selection, but after about 50 it may represent a more complete picture of the experience of medical practitioners. Table II includes all the "coronary" deaths in the population described in Table I. Columns 2, 4, and 6 give the number of deaths; columns 3, 5, and 7, in italics, the relevant rates.

TABLE II.—*Mortality from Coronary Heart Disease in Medical Practitioners, 1947–50. Men Aged 35–64 Inclusive. Average Annual Rates per Thousand*

| Ages Last Birthday (Years) (1) | Deaths in First Six Days of First Attack | | Deaths in First Month of First Attack | | All "Coronary" Deaths | |
|---|---|---|---|---|---|---|
| | No. (2) | Rate (3) | No. (4) | Rate (5) | No. (6) | Rate (7) |
| 35–39 .. | 1 | — | 1 | — | 2 | — |
| 40–44 .. | 9 | *0·8* | 5 | *0·9* | 5 | *0·9* |
| 45–49 .. | | | 6 | *1·0* | 8 | *1·3* |
| 50–54 .. | 14 | *2·1* | 5 | *1·2* | 8 | *2·0* |
| 55–59 .. | | | 12 | *4·3* | 13 | *4·7* |
| 60–64 .. | 5 | *3·1* | 9 | *5·5* | 12 | *7·4* |
| 35–64 incl. .. | 29 | *1·2* | 38 | *1·5* | 48 | *1·9* |
| 40–59 ,, .. | 23 | *1·4** | 28 | *1·7** | 34 | *2·0** |
| 40–64 ,, .. | 28 | *1·6** | 37 | *2·2** | 46 | *2·8** |

* *Standardized rates.*

For notes on ages, the Society's population, and standardized rates, see Table I.

Columns 4 and 5: Lengths of sick absences were calculated in years to 2 decimal places. Deaths in the first "month" were taken to be those occurring in 0·08, or less, of a year.

Columns 6 and 7: All deaths ascribed to coronary heart disease, as defined. The rates of column 7, Table II, may be compared with the American rates for 1938–42:

*Mortality From Coronary Heart Disease in Medical Practitioners of U.S.A.*

| Age | | Age | |
|---|---|---|---|
| 35-39 .. | 0.37 per 1,000 | 50-54 .. | 3.76 per 1,000 |
| 40-44 .. | 1.02 ,, ,, | 55-59 .. | 5.92 ,, ,, |
| 45-49 .. | 1.79 ,, ,, | 60-64 .. | 8.03 ,, ,, |

(Dublin and Spiegelman, 1947; Dublin, 1951).

Columns 2 and 3 deal with the deaths occurring quickly and referred to throughout this report as "sudden." The exact duration of the illness in the present series of "sudden" deaths cannot be specified. No benefit is paid for absence lasting less than seven days; and in these cases, since the question of a claim for sickness benefit does not arise, there is no obligation on the relatives or executors to state how long the illness lasted, or on the Society to ask for the information. Though it is known that in the majority the death occurred very quickly, it can only be stated of them all that they occurred within six days of the onset of illness causing incapacity to work. The "sudden" death rate, column 3, rises to 3.1 per 1,000 at 60–64 years of age. Columns 4 and 5 of Table II include all deaths occurring in the first month of the clinical disease; the rate rises to 5.5 at 60–64 years. Columns 6 and 7 represent all deaths certified as due to coronary heart disease in these practitioners (Fig. 1). This rate reaches 7.4 per 1,000 at 60–64 years.†

The main fact that emerges from Table II is the predominance of the first clinical attack, even as represented here only by the first month of absence from work, in the total mortality from coronary heart disease in these men under 65 years of age. About 80% of the mortality at 40–64 from coronary thrombosis, myocardial infarction, and their consequences, in 1947–50, occurred in the first month (cf. columns 5 and 7 of Table II). Sixty per cent. of the total coronary mortality rate represented the deaths occurring, as described, very quickly in the first few days of the first clinical attack—standardized rates of 1.6 per 1,000 in column 3 and 2.8 per 1,000 in column 7. (The Society's experience of 1940–6 was similar enough to that of 1947–50 for the deaths of the 11 years 1940–50 to be amalgamated in Fig. 2.) If this figure of 55 or 60% is at all typical of

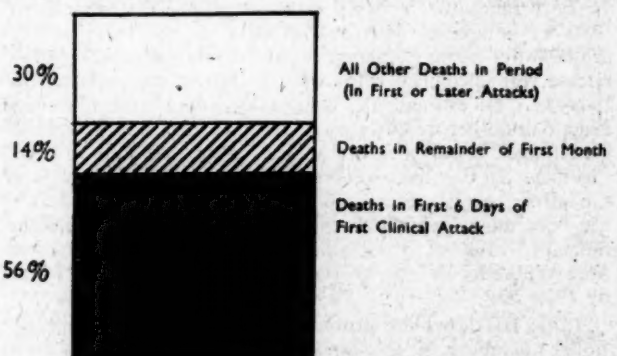
30%
14%
56%
All Other Deaths in Period (In First or Later Attacks)
Deaths in Remainder of First Month
Deaths in First 6 Days of First Clinical Attack

FIG. 2.—Deaths from coronary heart disease occurring in 1940–50. Male medical practitioners aged 40–64, inclusive. There were 104 coronary deaths in these members during the 11-year period.

what is happening in the general population in the coronary heart disease of middle age, it sharply focuses the gravity of the "acute" phases of this "chronic" disease, and points very clearly to the time of greatest need for therapy—the first few days of the first clinical attack of "coronary thrombosis." The size of this figure is interesting for two other reasons: it reinforces the conviction formed during the earlier investigation (Morris, 1951) that cardiac pathology cannot be studied properly to-day unless there is the closest relationship between the hospital and the coroner's court, especially if the hospital cannot, or does not, provide

†Gosse (1942) made a study of material from the same source, in which he paid attention to the mortality from coronary heart disease among the medical profession. In wartime conditions it was not possible for the Society to construct the considerable subsidiary recording system necessary to produce the results here published. In particular, the Society's normal records in some respects concern *policies* rather than *lives*, and many members have more than one policy. Much of the labour involved in the present inquiry has been spent in ensuring that information based on policies has been properly allocated to lives. It follows that the analyses in this paper cannot be compared directly with the broader figures given by Gosse, and no attempt has been made to do so.

facilities for the B(rought) I(n) D(ead). The size of this fraction may also explain some of the variation in the mortality rates reported in hospital series of "coronary thrombosis" (McCain, Kline, and Gilson, 1950; Tulloch and Gilchrist, 1950). If hospitals have an open door for all types of "emergencies" they are more likely to admit these men who will die in the first few days, with consequences in their statistics. Hospitals having a different admission policy may see few such patients, and show much "better" mortality rates, though the general practitioners and coroner's pathologists of the district may find the rates surprising.

Before leaving these morbidity and mortality figures it may be mentioned that there was no particular concentration of first attacks or of deaths in any part of the year, though the April–June quarter had the lowest numbers.

### The Individual Practitioner's Chances

What are the chances that members of the Society will develop coronary heart disease, and die of it, should current attack and mortality rates continue? The numbers in the present inquiry are small, but the rates (Tables I and II) progress with sufficient regularity to justify an attempt to answer these questions. Appendix 1 sets out the details of the methods adopted: here it need only be said that a "life table" approach was made. To estimate the chances of *getting* the disease before age $x$, the rates of first clinical attack at different ages under $x$ are used, and the resulting numbers attacked at each age are summed. These rates, however, are not quite the same as in Table I, because the present calculations are concerned only with the incidence among those who have not yet had the disease, and are thus "eligible" for a first attack, whereas Table I deals with all who are alive at particular ages. Moreover, there is some evidence in the four years' material of an increase in the occurrence (or recognition) of non-fatal first attacks in 1949–50, compared with 1947–8, and, as the basis of the probabilities being estimated is the *current* frequency of the disease, the incidence rates were therefore recalculated for 1949–50. To estimate the chances of *dying* from coronary heart disease before any particular age $x$, the chances at individual ages under $x$ are similarly calculated and brought together. In this instance the "eligibles" are all those who are alive, because those who have had the disease before are very much exposed to the risk of dying of it; and the mortality rates of Table II were used because there was little difference in the mortality experience of 1947–8 and of 1949–50.

Table III shows the probability that these medical-practitioner members of the Society, not as yet suffering from clinical coronary heart disease, will be attacked before particular ages. If present rates of incidence continue, it can be seen by reading horizontally along the bold figures that, of 1,000 men aged 35, three may be expected to develop the disease in the next five years—that is, before they are 40, another seven by 45, and so on. Of 1,000 aged 60, 90 will be likely to get it in the next five years—that is, before they are 65. The cumulative "chances" up to age 65 are shown in the last column of Table III, and indicate, for example, that, of 1,000 at 35 or 40, 200, or one in five, are likely to get the clinical disease before age 65; and the proportion is little different at 45. Since the probability of getting the clinical disease *under* 35 years of age is extremely small, it may be said, roughly speaking, that the chances at 35 represent the chances from the beginning, and that they continue about the same till 45—that is, these medical practitioners under 45 years of age who have not so far been attacked by coronary heart disease have approximately a 20% chance of being attacked before they reach 65. The estimates for intermediate periods can easily be read from Table III—for example, of 1,000 at 50 years of age the chances are that 110 will get coronary heart disease before 60.

TABLE III.—*Chance of Getting Coronary Heart Disease*

The number of male medical practitioners who, not having previously been clinically attacked by it, would get coronary heart disease before reaching certain ages.

| Age $x$ (Years) | Of 1,000 Men Aged $x$, the Number Indicated Below Would Get Clinical Coronary Heart Disease Before Age | | | | | |
|---|---|---|---|---|---|---|
| | 40 | 45 | 50 | 55 | 60 | 65 |
| **35** | **3** | **10** | **27** | **67** | **130** | **200** |
| 40 | | 7 | 24 | 65 | 129 | 200 |
| 45 | | | 18 | 59 | 124 | 196 |
| 50 | | | | 43 | 110 | 184 |
| 55 | | | | | 72 | 152 |
| 60 | | | | | | 90 |

*Coronary heart disease* refers to the clinical manifestations of "coronary thrombosis," angina pectoris, etc., as defined.

Rates of first attack used in calculating the table are based on the Society's experience in 1949–50; rates of death are based on data for 1947–50. The table is constructed on the assumption that both these rates will continue indefinitely.

The "population" is as described in Table I, but excludes in each age group those who have already been attacked by coronary heart disease.

Technical details may be found in Appendix 1.

Table IV provides similar estimates for the chances of dying. It shows, for instance, reading again along the bold figures, that, of 1,000 men alive at 35, 75, or 7.5%, will be likely to die of coronary heart disease before they reach 65, should current death rates in the Society continue. Of 1,000 at 60, 38 may be expected so to die before 65.

TABLE IV.—*Chance of Dying from Coronary Heart Disease*

The number of male medical practitioners who would die of coronary heart disease before reaching certain ages.

| Age $x$ (Years) | Of 1,000 Men Aged $x$, the Number Indicated Below Would Die of Coronary Heart Disease Before Age— | | | | | |
|---|---|---|---|---|---|---|
| | 40 | 45 | 50 | 55 | 60 | 65 |
| **35** | **3** | **6** | **12** | **23** | **42** | **75** |
| 40 | | 3 | 9 | 20 | 39 | 73 |
| 45 | | | 6 | 17 | 37 | 71 |
| 50 | | | | 11 | 31 | 66 |
| 55 | | | | | 20 | 56 |
| 60 | | | | | | 38 |

Rates of mortality used in calculating the table are based on the Society's experience in 1947–50, and the table is constructed on the assumption that these rates will continue indefinitely.

The "population" is as described in Table I.

Technical details may be found in Appendix 1.

Fig. 3 summarizes both tables in terms of a hypothetical 15 male practitioners aged 35.

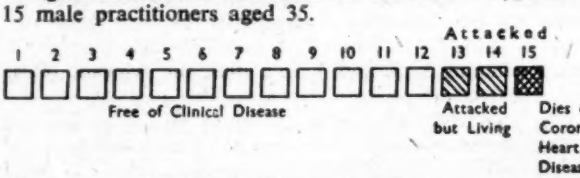


FIG. 3.—The individual practitioner's chances. Of 15 practitioners aged 35, 12 can expect to be free of clinical coronary heart disease up to the age of 65, two to suffer from the disease without dying, and one to die of it before that age. These figures are based on the current experience of the members studied. They are less precise than the figures in Tables III and IV, from which they are derived.

### Prevalence of Coronary Heart Disease

The probability among these medical practitioners of developing coronary heart disease before 60 years of age is about one in eight, before 65 about one in five; but that does not mean that 12 to 20% of them at 60–64 years of age will have the disease: many will die beforehand. It is worth trying to get some idea of the *prevalence* of coronary heart disease in these medical practitioners, of how many of them are *affected* during a particular time, whether by a first attack of the disease or any later manifestation of it. No corresponding figures seem to be available for any other population group. The following methods were used.

Consider, for simplicity, the population born, say, in 1890, who are alive at some time during 1950. This population could be divided as follows:

(*a*) Men who have an attack starting in 1950 which is the first clinical manifestation of coronary heart disease.

(*b*) Men who have an attack starting in 1950 which is a second or later clinical episode of coronary heart disease, but who do not have a first attack in 1950.

(*c*) Men who had an attack before the beginning of 1950, who continue to be incapacitated for part or all of that year, but have no attack starting in 1950.

(*d*) Men who have had an attack of coronary heart disease in an earlier year but, having returned to work, do not have an attack starting in 1950.

(*e*) Men who have never been attacked by the clinical disease, and do not suffer from it during 1950.

(In the present paper, of course, "attack" means "absence" for at least seven days or death.) These classes are mutually exclusive and make up the total of these medical practitioners or, indeed, of any "population." Class *a*, it will be remembered, gives the *incidence* and has already been considered. Class *b* together with Class *a* gives what may be called a *total attack rate* of all persons attacked in a period. This is not a particularly interesting statistic and has not been considered in this report.† Classes *a*, *b*, and *c*

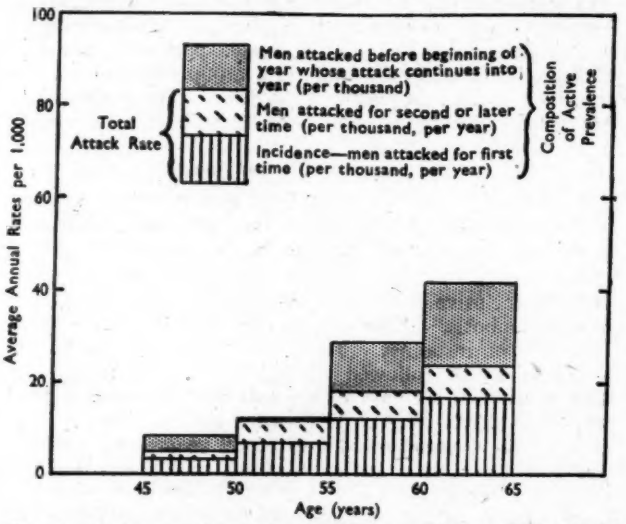
Men attacked before beginning of year whose attack continues into year (per thousand)
Men attacked for second or later time (per thousand, per year)
Incidence—men attacked for first time (per thousand, per year)
Total Attack Rate
Composition of Active Prevalence
Average Annual Rates per 1,000
Age (years)

FIG. 4.—Active prevalence of coronary heart disease, 1950. Male medical practitioners aged 45–64 inclusive. The active prevalence figures refer to 1950 only (Table V); the total attack rates are averages for 1949 and 1950 (see text), and the incidence rates for 1947–50 (Table I).

together, in the present material, form the total number of men disabled from work during the year for as much as a week, or who die—a figure that may be called the "*active prevalence*" of the disease. Columns 2 and 3 of Table V describe this in the present population, in the year 1950.

TABLE V.—*Prevalence of Coronary Heart Disease in Medical Practitioners, 1950. Men Aged 35–64 Inclusive. Average Annual Rates per Thousand*

| Ages Last Birthday (Years) (1) | "Active Prevalence" | | "Latent" Disease | | "Total Prevalence" per 1,000 (6) |
|---|---|---|---|---|---|
| | No. (2) | Rate (3) | No. (4) | Rate (5) | |
| 35–39 | 2 | — | — | — | — |
| 40–44 | 2 | — | 5 | 3·7 | 5·2 |
| 45–49 | 13 | 8·3 | 13 | 8·3 | 16·7 |
| 50–54 | 14 | 11·9 | 20 | 17·0 | 28·9 |
| 55–59 | 21 | 28·7 | 19 | 26·0 | 54·7 |
| 60–64 | 19 | 41·6 | 22 | 48·1 | 89·7 |
| 35–64 incl. | 71 | 11·1 | 79 | 12·4 | 23·5 |
| 40–59 " | 50 | 10·6* | 57 | 12·4* | 23·5* |
| 40–64 " | 69 | 15·2* | 79 | 17·6* | 33·2* |

* *Standardized rates.*

"Active prevalence" = men disabled from work for at least seven days because of coronary heart disease, during any part of 1950. (The sum of Classes *a*, *b*, and *c* in the text.)

"Latent" disease = men who had an attack of coronary heart disease during 1940–9 inclusive, but who were not disabled from work for at least seven days on that account during 1950. (Class *d* in the text.)

"Total prevalence" = the sum of "active prevalence" and "latent" disease.

The mean of the counts of the relevant part of the Society's membership as on January 1, 1950, and January 1, 1951, was used for the population.

†The relevant rates per 1,000 in 1949–50 were: at 35–39 years, 0.9; at 40–44, 1.1; at 45–49, 5.1; at 50–54, 11.8; at 55–59, 18.1; and at 60–64 years, 23.7 (see Fig. 4.)

The "active prevalence" rate climbs with age to about 40 per 1,000, or 4% of all alive at 60–64 years (Fig. 4).

This "active prevalence" rate, however, tells only part of the story, as can be seen from Fig. 1, where the total coronary mortality (column 7 of Table II) is plotted against the incidence of new cases (column 4 of Table I). It is at once obvious that there must be a steady and substantial build up of non-fatal cases which is seriously understated in the "active prevalence." In clinical terms it is clear why this rate is an inadequate picture of the total burden of the disease. The "active prevalence" measures the disability to work; but many who have an attack of "coronary thrombosis" return to work (it may be very modified work), and thus may not appear in a complete disability rate in any particular year, however much their mode of life is otherwise changed by the disease. These are the Class *d* cases, and quite a number of them may in fact have manageable angina of effort. What is required, therefore, is a measure of all who have been attacked by coronary heart disease, have survived it, and in whom the disease is "latent," though *potentially* it can become very "active" at any time and lead to complete disability to work—Class *d*.

The records were therefore searched for all men born in 1885–1914 (corresponding to ages 35–64 years in 1950) who had suffered from coronary heart disease in the period 1940–9 and who were alive in 1950, but not absent from work in that year on account of it, and thus included in the "active prevalence." The present material is particularly suitable for this kind of study because men can be observed till they reach 60 or 65: they cannot be written off the books for ill-health (as in a factory), nor do they lapse from follow-up study (as may happen in a cardiac clinic), and it is rare for any man to give up all his non-cancellable sickness policies.

The result of this search was to add to the "active prevalence" (column 3 of Table V) a "latent" prevalence of less active disease (column 5) slightly larger than the "active prevalence" itself, and reaching almost 50 per 1,000, or 5%, at 60–64. These figures, moreover, may slightly underestimate the true position, as the experience before 1940 was not examined, and this might have yielded a few further survivors to add to the totals with "*latent*" *disease* in 1950.

The "active prevalence" and "latent" disease can be added together to give an estimate of the total clinical prevalence, or the cumulative burden of the disease, in this population group (Fig. 5). The "*total prevalence*" of coronary heart disease in these medical practitioners in 1950, at 50–54 years of age, was about 3% in all. In those

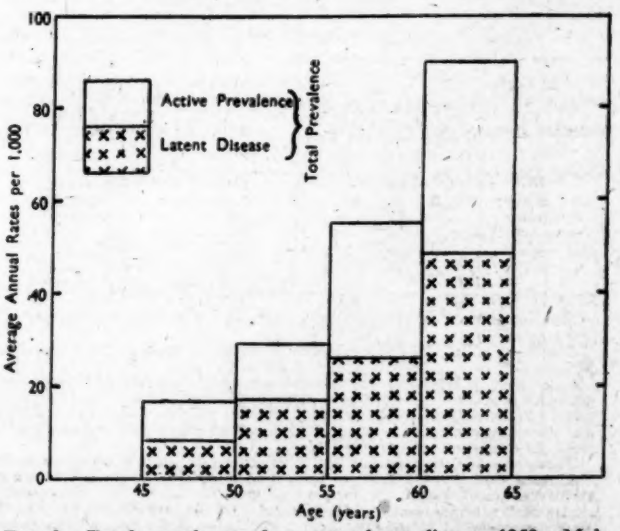


FIG. 5.—Total prevalence of coronary heart disease, 1950. Male medical practitioners aged 45–64, inclusive (see Table V). Latent disease = men attacked in a previous year who are not absent on account of the disease during the year.

surviving to 60–64 years of age the "total prevalence" was about 9%. In the light of such figures terms like "epidemic" may be justified.

### Prognosis

Up to this the main problem has been, What proportion of men are attacked by coronary heart disease?—for example, as in Tables I, III, and V. Attention will now be turned to prognosis, and the questions that will be asked can be expressed as, What happens to men so attacked? The present series of questions, that is, deals only with the *cases* of the disease, and not with the *population* in which these occur; the approach will be the usual one of the clinician, not that of the epidemiologist. It is therefore possible to make use of cases occurring in the Society before 1947, even though details of the membership at different ages in those years are not available in a form suitable for calculation of rates. The records of first attacks of coronary heart disease in 1940–6 were therefore extracted, and the information was added to that already available for 1947–50. (Medical papers relating to most of the deaths before the war were lost by enemy action in 1940.) The Society's experience is a useful basis for studies of prognosis because, as stated, regular contact is kept with all members until they reach the age limit or die. On the other hand, the fact that the data deal only with men under 65 years of age is a serious limitation in discussing the prognosis for life, though not of course for work. The present inquiry was stopped on June 30, 1951, an arbitrary date on which it was convenient to cease observation of the men. Appendix 2 describes the methods adopted in drawing up Tables VI and VII.

TABLE VI.—*Prognosis for Life and Death in "Coronary Thrombosis." Experience of Medical Practitioners from the Onset of the First Clinical Attack, 1940–51. Men Aged 40–64 Inclusive*

(A) Percentage of men dying and surviving at various periods from onset of first attack.

| Time | First 6 Days | Rest of 1st Month | Rest of 1st Year | 2nd and 3rd Years | 4th and 5th Years | 6th and 7th Years |
|---|---|---|---|---|---|---|
| Dying in period | 30 | 8 | 2 | 6 | 3 | 3 |
| Cumulative mortality* | 30 | 38 | 40 | 46 | 49 | 52 |
| Surviving period | 70 | 62 | 60 | 54 | 51 | 48 |

* Up to end of period specified.

(B) Percentage of men dying and surviving, by age.

| Ages Last Birthday (Years) | Dying in First 6 Days | Dying in 1st Month | Dying in First 3 Years | Surviving First 3 Years |
|---|---|---|---|---|
| 40–49 | 29 | 33 | 39 | 61 |
| 50–64 | 31 | 40 | 50 | 50 |
| 40–64 incl. | 30 | 38 | 46 | 54 |

These tables are based on the experience of 192 medical practitioners, who had a first attack of "coronary thrombosis" between January 1, 1940, and December 31, 1950. Observation was continued till June 30, 1951, except for men who died or whose policies terminated before. The total number followed in the fifth year was 46, and in the seventh, 26. In the third year there were 35 men aged 40–49, and 39 aged 50–64 inclusive. Men whose first presentation of coronary heart disease was not as an attack of "coronary thrombosis" were excluded from these tables. Details of the methods of calculation used may be found in Appendix 2.

TABLE VII.—*Absence from Work on Account of Coronary Heart Disease After the First Attack of "Coronary Thrombosis." Experience of Medical Practitioners who Survived Their First Illness, 1940–51. Men Aged 40–64, Inclusive*

| (A) Second Absences | | (B) Working Time Lost | |
|---|---|---|---|
| No. of Years After the End of First Attack = *t* | % Survivors of First Attack Absent from Work Again, Because of Coronary Heart Disease, Within *t* Years | No. of Year After the End of the First Attack | Average Time Lost per Man Because of Coronary Heart Disease During the Year |
| (1) | (2) | (3) | (4) |
| 1 | 14% | 1st | 0·6 month |
| 2 | 30½ | 2nd | 1·1 months |
| 3 | 33½ | 3rd | 0·9 month |
| 4 | 35½ | 4th | 0·8 ,, |
| 5 | 45½ | 5th | 0·8 ,, |
| 6 | 49½ | 6th | 0·9 ,, |

The first absence for "coronary thrombosis" was taken to represent the first attack.

Columns 1 and 3: The date of return to work was taken as the "end of the first attack."

Column 4: "Time lost" is calculated from the number of days of total disability to work. No attempt is made to estimate the number of working hours lost, by distinguishing working days from Sundays and public holidays; and no allowance could be made for periods of absence lasting less than a week, or for periods of part-time work.

There were 107 survivors of the first attack. For Table A observation was continued until June 30, 1951, or until the second absence (or death without further absence), except for men whose policies terminated. The number under observation in each year diminishes from the original 107 to 35 in the fourth year and 21 in the sixth year. For Table B the only difference was that observation did not cease with a second absence. The numbers under observation in any year are therefore slightly greater.

Details of the methods used in calculating the proportions and averages in these tables are given in Appendix 2.

### Prognosis in "Coronary Thrombosis"

Without arguing the unitary pathology and, in particular, functional pathology of coronary heart disease associated with atheroma, this discussion falls naturally into two parts—the prognosis in "coronary thrombosis" (myocardial or cardiac infarction), and that in angina pectoris (angina of effort). The level of diagnosis in a group like this is probably high enough to justify the division. The prognosis in "coronary thrombosis" will be dealt with first.

#### Prognosis for Life and Death

It is already clear from Table II that the crucial period for life and death, at any rate in the ages being considered, is the first few days of the first clinical attack.

*First Days of First Clinical Attack.*—In the 11 years 1940–50 inclusive there were 192 first diagnoses of "coronary thrombosis," as defined, in men aged 40–64 who held the relevant insurance. These men may be regarded as a cohort to be followed through. The proportion who died in the first six days of the first clinical attack (Fig. 6) was 30% (Table VI,A). It varied a little with age—from 28% at 40–44 years, to 34% at 55–59 years—and the relative stability of this fraction through middle age was not altogether expected. There was no change in the fraction from 1940–6 to 1947–8; in 1949–50 it was rather lower, which may be an indication, possibly, of an increasing diagnosis of less classical and milder cases.

*Rest of the First Month.*—The next column in Table VI,A shows the mortality in the remainder of the first month. It is still heavy—8% of the original number of men (and 11% of those surviving)—but not nearly so heavy as in the first few days (Fig. 6). In total, therefore, about 38% of these medical practitioners who were first attacked by coronary heart disease in the form of a "coronary thrombosis" in 1940–50 died in the first month of their illness; about 62% survived the first month. It is not known whether any of these men received anticoagulant treatment. It is unlikely that the deaths in the first few days could have been much affected by this form of therapy, and the number of deaths in the remainder of the first month, both in 1940–6 (when there must have been little such treatment) and in 1947–50 (when there may have been much), is impossibly small for

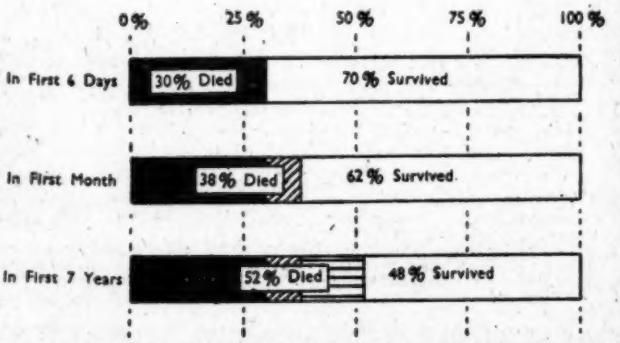


FIG. 6.—Prognosis for life and death in "coronary thrombosis." Experience of medical practitioners from the onset of the first clinical attack, 1940–51. Men aged 40–64 inclusive (see Table VI,A).

any comparisons; all the more since other changes in the same period, such as an improvement in diagnosis, need to be considered.

Attention is now turned to the survivors of the first month, whose passage, it may be said at once, is decidedly less stormy than heretofore.

*In Following Years.*—In the remaining 11 months of the first year another 2% of the original cohort died from coronary heart disease, or its complications, and this figure is typical of the annual experience in the next six years. Table VI,A gives some detail—in the second and third years from the onset, for example, a further 6% died, making a total mortality up to the end of the three years of 46% (30%+8%+2%+6%); 54% of the original cohort who could be followed survived the three years.

*In Younger and Older Men.*—Table VI,B gives a simple analysis by age. There is little difference in the outlook during the first six days; thereafter, for the three years which can be adequately followed, the prognosis seems to be better in the younger men. By the end of the third year there is a difference of 11%, with 61% of the men aged 40–49 at onset of first attack of "coronary thrombosis" surviving, but 50% of those who were aged 50–64 years. This conforms with the general experience on the deterioration of prognosis with age (Friedberg, 1949; Eckerström, 1951). However, the numbers are too small to exclude the possibility that we may be dealing with a chance effect.*

### Prognosis for Further Sick Absence

Analysis of the further absences from work on account of coronary heart disease among those who have survived their first illness is worth while in clinical and social terms, if not in pathological terms. The frequency of such further absences is high in the two years following the return to work, Table VII,A, with 30% of the men having a second bout of absence. The cumulative proportion of those who have had a further sick absence for coronary heart disease, or its complications, reaches about 50% by the end of the sixth year. No differences with age were discernible in these further absences—that is, the younger men seemed to have as many second absences as the older, though, as was noted before, their mortality on the whole may be less. Numbers, however, are small in the different age groups.

### Prognosis for Work

The point of view here is rather different: in the formulation of any policy for, say, the future employment of men with coronary heart disease it is necessary to know their capacities as well as their needs. In practical terms, how much time may men who have survived their first attack (as reckoned by their return to work after the first illness) expect to lose in the years thereafter? The present material contains some evidence on this, and its restriction to ages under 65 does not matter so much. The length of each absence is known, and it is therefore possible to calculate the average amount of time lost by each man per year. It is of course likely that some of the men who did not claim sickness benefit were in fact partially disabled and on less than full duty; but this partial disablement cannot be included in Table VII,B. The figures in that table indicate that, on average, the survivors of the first clinical attack of "coronary thrombosis" missed only about one month's work in each of the next six years because of coronary heart disease (Fig. 7). Some of these men, as shown above, died during the years following the first attack; they are included in the present account until the year in which they went absent because of their final illness. More precisely, therefore, Table VII,B indicates, for instance, that the men who survived *to* the beginning of the fifth year from the end of the first attack lost an average of 0.8 month (or about four weeks) *in* the fifth year because of coronary heart disease.

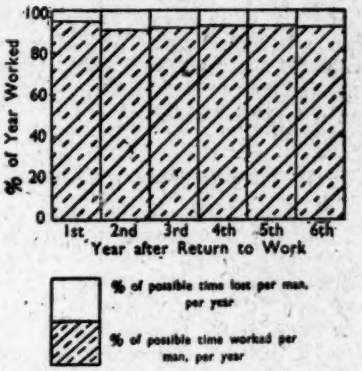


FIG. 7.—Prognosis for work. Experience of medical practitioners who survived their first illness, 1940–51. Men aged 40–64 inclusive. Sick absence for other than coronary heart disease, its complications and consequences, has been ignored (see Table VII,B).

### Prognosis in Angina Pectoris

There were 42 first attacks of angina pectoris (or "of effort") during 1940–50 which were ascribed to coronary atheroma—or in which no underlying pathology was stated—in comparison with the 192 first attacks of "coronary thrombosis"; (and nine, which have been ignored, of "coronary insufficiency" and the like). Presentation with angina pectoris became commoner with age (Fig. 10). The first absence in these cases of angina lasted on average for about three months compared with about five months for "coronary thrombosis." Later absences of men who initially were absent with angina were increasingly ascribed to "coronary thrombosis," of course, and lasted longer.

With such small numbers examination of prognosis is less profitable, but one fact did emerge clearly: the outlook for life among the cases first presenting as angina pectoris is

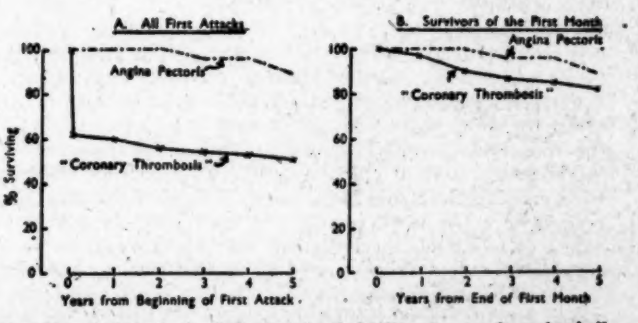


FIG. 8.—Prognosis for life and death in "coronary thrombosis" and angina pectoris, 1940–51. Male medical practitioners aged 40–64, inclusive.

*Prognosis in the First Attack and Second Attack.*—Clinically it is even more important to know the outlook in the various attacks of "coronary thrombosis." The present material, however, is not very suitable for this type of analysis. These records are only of sick absence and death, and a *further* attack supervening during the convalescence from a *first*, for example, would not necessarily be recorded separately, if the patient had not yet returned to work from the first illness. Nevertheless, figures for the first month do probably refer to the first attack of "coronary thrombosis," and probably include most of the deaths *in* the first attack. The only indication that can be given of the mortality in second attacks is the experience in second *absences*. Many of these very likely refer to second attacks; but a few may refer to even later attacks (if more than one attack was included in the first absence); and a few may be due to relapses of the first attack; or complications and cardiac failure; or to external factors aggravating the cardiac condition. Following are the relevant figures; they show the trend with age, particularly after the first week:

| Ages Last Birthday (Years) | Experience | First Attack/Absence: First 6 Days | First Attack/Absence: Rest of 1st Month | Second Absence (and ? Attack) |
|---|---|---|---|---|
| 40–49 incl. | No. of men followed through .. .. | 63 | 45 | 16 |
| | No. dying .. .. | 18 | 3 | 2 |
| 50–64 incl. | No. of men followed through .. .. | 129 | 89 | 17 |
| | No. dying .. .. | 40 | 12 | 6 |

Third absences are too few for analysis.

very different from that of the men first presenting as "coronary thrombosis." In angina it is similar, in the five years that can be studied, to the prognosis in men who have *survived* their first critical month of "coronary thrombosis" (Fig. 8). About 11% of the men first presenting with angina pectoris died in the next five years. About 16% of those who survived the first month of "coronary thrombosis" died in the next five years, but this figure has to be added to the 38% who had already died *in* the first month. In terms of a "severity" spectrum of the natural history of coronary heart disease, therefore, the more slowly developing, relatively benign, cases of angina pectoris seem to be at one end, the "sudden" deaths from "coronary thrombosis" as first clinical manifestation, at the other, with a wide range of severity between.

### Summing-up of Prognosis in Middle Age

If the figures in this series are at all representative, "coronary thrombosis," as regards its mortality, belongs to the most "acute" diseases rather than to the "chronic" diseases. It is clearly unwise to consider the prognosis for life of men first presenting with "coronary thrombosis" except in terms of the first days of the first attack (30% mortality in six days, in this series), the rest of the first month (8% mortality), and the years thereafter (about 2% of the original cohort died annually in each of the next seven years) (Fig. 9). About 80% of those who survived the first month were

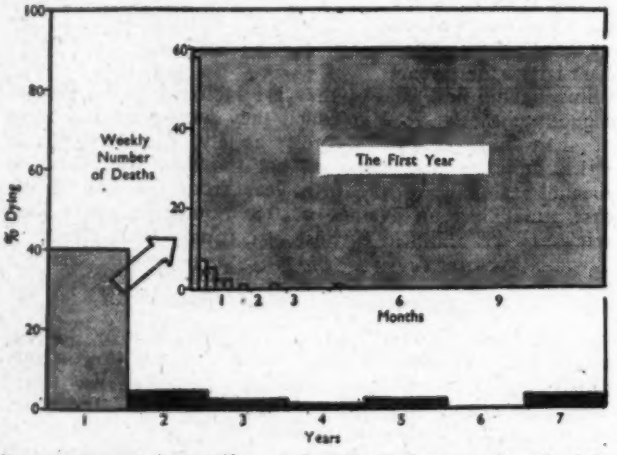


FIG. 9.—Prognosis for life and death in "coronary thrombosis." Experience of medical practitioners from the onset of the first clinical attack, 1940-51. Men aged 40-64 inclusive. Distribution of deaths by time from onset of first attack (see Table VI,A).

*Number of Deaths in Each Week in the First Year*

| Week | Deaths | Week | Deaths |
|---|---|---|---|
| 1 | 58 | 7 | 1 |
| 2 | 7 | 8–10 | 0 |
| 3 | 5 | 11 | 1 |
| 4 | 2 | 12–18 | 0 |
| 5 | 2 | 19 | 1 |
| 6 | 0 | 20–52 | 0 |

alive at the end of five years. This improvement after the first month was due not so much to mortality in later attacks being less than in the first—though this also may be so—as to the fact that a second attack does not necessarily occur at all for some years. On the present evidence, about half the men who survived their first illness and returned to work did not have as much as a week's further sick absence for coronary heart disease in the six years following. In consequence the work record, for whole-time or part-time work, of men who survived their first attack is good: they may each lose on average only about four weeks a year for coronary heart disease in the six following years. The prognosis for life of men first presenting as angina pectoris seems to be better than that of men first presenting as "coronary thrombosis." Indeed, they seem to miss the early crises altogether, and their outlook, for a few years at any rate, is similar to that of the men with "coronary thrombosis" who have survived the first terrible month.*

This discussion of prognosis deals only, of course, with middle-aged medical practitioners. The evolution of coronary heart disease in the elderly may be quite different.

### Mode of Presentation of Coronary Heart Disease

Fig. 10 illustrates the fraction at each age that the cases of "sudden" death form of all presenting coronary heart disease in these medical practitioners. It can now be stated

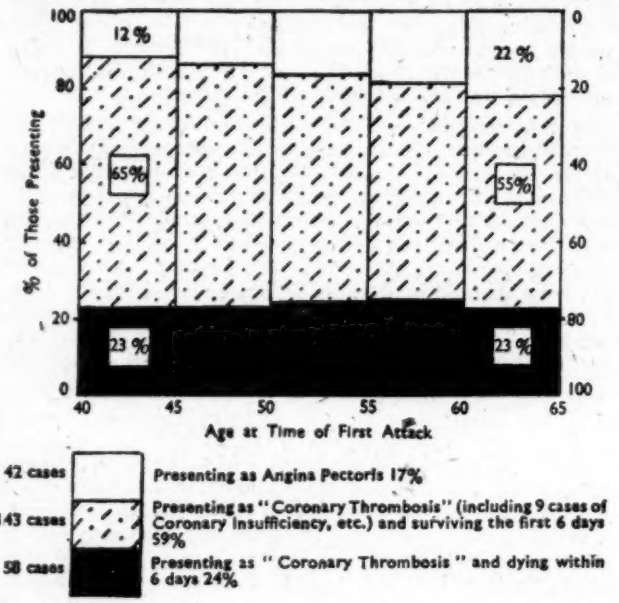
% of Those Presenting
12 %
22 %
65%
55%
23 %
23 %
Age at Time of First Attack
42 cases
Presenting as Angina Pectoris 17%
143 cases
Presenting as "Coronary Thrombosis" (including 9 cases of Coronary Insufficiency, etc.) and surviving the first 6 days 59%
58 cases
Presenting as "Coronary Thrombosis" and dying within 6 days 24%

FIG. 10.—Mode of presentation of coronary heart disease, 1940–50. Male medical practitioners aged 40–64, inclusive.

in round numbers that at 40–44 years of age coronary heart disease presents as "sudden" death (within six days) in about a quarter of the cases, as less severe "coronary thrombosis" in about 60%, and as angina pectoris in somewhat over 10%. At 60–64 years of age the fractions are again about a quarter as "sudden" deaths, somewhat over 50% as not so severe "coronary thrombosis," and somewhat over 20% as angina pectoris. These three manifestations

*It is perhaps worth while to stress here the difference between two fractions which are discussed in this paper. The first, mentioned previously under "Mortality Rates," gives the proportion of deaths within six days of the onset of the first attack—that is, "sudden" deaths—formed of *all deaths* from coronary heart disease (whenever these deaths occurred, whether in the first or later attacks). At *ages 40–64*, in the 11-year period 1940–50, this fraction was 56%. The second fraction, discussed in the present section on prognosis, is the proportion of "sudden" deaths among all the *first attacks* of "coronary thrombosis." In the same period, and in men of the same age, this second fraction was 30%. Since it is probably true that most people who have coronary heart disease, especially "coronary thrombosis," will eventually die of it, there is at first sight an apparent contradiction here. Closer examination shows, however, that the first fraction—that is, of the observed deaths—depends on the ages studied; whereas the second—that is, of first attacks—does not appear to vary much, at least within the age range 40–64. During 1940–50 "sudden" deaths, as said, formed 56% of all "coronary" deaths: but this 56% was made up of 62% at ages 40–49, 58% at 50–59, and 44% at 60–64 years. It is easy to account for this variation. At early ages there will be few people who have been previously attacked by the disease and who are therefore likely to die at long intervals after the first attack. The fraction, therefore, falls with age, and quite possibly at greater ages than 65 it will be even lower than 44%. The fraction which "sudden" deaths form of all coronary deaths *at all ages* is thus likely to be considerably lower than 56%, and the contradiction between this figure and that of 30% may well be a result of the particular age range studied, and the limited period under observation. However that may be, the first and higher fraction of 56%, applying as it does to men aged 40–64, brings into prominence the fact that more than half of the deaths that do occur in middle age, when the problems of the disease are at their most serious, are in fact "sudden."

accounted for all but nine of the 243 "first presentations" of coronary heart disease in the years 1940–50, inclusive. The nine were variously described as "coronary insufficiency," etc.

## PART II: THE DISEASE IN GENERAL PRACTITIONERS AND IN OTHER DOCTORS

In a social medical inquiry, concerned eventually with possible relationships between health and ways of living, "medical practice" is rather a wide social category to be assumed homogeneous in its experience of a disease. The general practitioner, the consultant, and the medical officer of health may lead professional lives at least as disparate as many "occupations" usually distinguished in this type of study. The practical difficulty is how, without personal inquiry, to decide which of a large group of doctors are G.P.s, consultants, etc., so that the experience of different sections of the profession can be compared. The only readily available source of information on what doctors do is the *Medical Directory*; and study of its contents showed that it is practicable to classify doctors into the following broad categories:

*Category 1.*—This comprises those who, it may reasonably be presumed, are full-time *general practitioners* (or almost so)—doctors with no appointments and merely a name and address in the *Directory*; or with certain types of appointment, such as Post Office medical officer or appointed factory doctor, and no other appointments.

*Category 2.*—This, so far as can be determined, is composed of full-time clinical *consultants/specialists*—men on the staff of teaching hospitals, or certain other major hospitals; and men with at least two specialist appointments at non-teaching hospitals (in certain cases having particular diplomas). Any indications of general practice, such as the mention of a partnership, would exclude a man from this category.

*Category 3.*—An intermediate group, fairly well defined outside the university centres, of general-practitioner specialists—that is, men who are both; who are in general practice and also do some specialist work, usually having one appointment, such as surgeon or physician (but not "clinical assistant" or "medical officer") at the local non-teaching hospital; doctors recorded as being merely "on the staff" of cottage hospitals are not included. Men in this category will be referred to as *G.P.-Specialists* in contrast to full-time general practitioners (Category 1), and full-time consultants/specialists (Category 2).

*Category 4.*—A number of small groups of various full-time *public officials*—medical officers of health, doctors in Government departments, regular officers in the armed Forces; and to these may be added a small number of full-time staff of mental and fever hospitals and tuberculosis sanatoria who are not of consultant status; and men holding academic non-clinical posts.

*Category 5.*—Retired practitioners; those of "address unknown"; and those who could not be classified.

This seemed a useful grouping, and it was decided to apply it in the next stage of the inquiry—an attempt to find whether any of the major sections of the profession, as these were represented in the Society, had more or less coronary heart disease than any other. There is good reason to believe that there is a high level of diagnosis among all the members, and that in a condition of such duration and gravity extraneous factors that might affect the sick-absence rates for minor ill-health will not apply. The attempt necessarily involved three steps: (1) to determine the total number in the various categories among the relevant members of the Society, at different ages, by sampling; (2) to identify the occupational category in each recorded case of coronary heart disease; and (3) to calculate the incidence of the disease in the various categories. It was decided to make a pilot test of the experience of 1949 and 1950.

Ten per cent. of the relevant membership at each year of age over 45 was taken (a strictly random sample using random sampling numbers); and the "first attacks" of 1949–50 in members aged 45–64 were shuffled in with this sample. First attacks only were included because men may change their jobs on account of their health, and if the possible effects of work on disease are the object of study, not the possible effects of disease on the work that men do, first attacks are likely to give the most reliable information. The cases were included with the sample to preserve the ignorance of the unit's staff of individual identity, and thus to eliminate any possible bias, as well as to preserve the confidential nature of medical records. All these persons—the 10% sample plus those attacked for the first time—were then marked in the *Medical Directory*. The two authors of this report who were ignorant which of the names represented a man with a first attack and which did not independently categorized all the names, discussing together and agreeing on the doubtful ones; there was no particular difficulty in allocation, and the great majority sorted themselves at once. The third author, who held the key to the names, then placed each person appropriately, by age and category, as a "first attack" or as a member of the sample population (as a matter of fact four "first attacks" were also drawn in the sample). Finally, rates of incidence of coronary heart disease were calculated for the various categories, on the assumption that the occupations in the whole of the relevant membership were distributed in the same way as those in the sample. The results, which were surprising, are summarized in Table VIII, for the general-practitioner group and the rest.

TABLE VIII

| Findings | Category 1 General Practitioners | Category 2+3+4+5 The Rest |
|---|---|---|
| No. of first attacks of coronary heart disease in 1949–50 at 45–64 years | 46 | 21 |
| No. in 10% sample of "population" | 213 | 197 |
| Annual incidence per 1,000 at 45–64 years (standardized for age) .. | 11·8 | 6·2 |
| Standard error of this rate .. .. | ±2·1 | ±1·5 |

Since it is unlikely that there is much difference in the level of diagnosis among different sections of the profession, or in the extent to which they would claim benefits in such serious and prolonged illness, these figures were suggestive of a true excess among the general practitioners. Critical examination of the data failed to upset this trend, which was evident in all five-year age groups, in each of the major geographical regions of the United Kingdom, and when the general practitioners were compared with each of the other categories taken by itself—all evidence of that "internal consistency" of the material so important in dealing with small numbers. Various manipulations had no effect—for example, the reallocation of "marginal" occupations among the various categories, the exclusion of all who were classified as general practitioners on *negative* grounds (that is, because they had no more than their names and addresses in the *Directory*), and the separation of fatal from non-fatal attacks. It did appear that the members designated as full-time general practitioners had a higher incidence than the other members of the Society.*

There are three major possible sources of weakness in the above trial: (1) the limitations of sampling; (2) the difficulties, and possibly the "fluke" effect of the small number of attacks, whereby one or two might materially alter the rates; and (3) the allotment of occupation on the basis of the *Medical Directory*. These weaknesses had to be dealt with so far as possible, and this is what we did. The first problem was met by drawing a further, and quite separate, 10% sample of the relevant membership; the occupational

*It may be mentioned that there was no material difference in the *ages* at which the general-practitioner members and the men in the other categories took out their first immediate benefit policies with the Society. The average age of this among the "*cases*" of coronary heart disease was 34.6 years in the general practitioners and 32.6 in the remainder; among the "sample" it was 32.6 years in the general practitioners and 31.4 in the others.

distribution of this sample was very similar to that of the first. The number of cases of coronary heart disease was increased by including the first attacks of 1947–8. (A further "trial" was then carried out by shuffling these 1947–8 cases in with the *new* sample, and the results were similar to those tabulated above.) The third problem, the allotment to type of work, was more troublesome. It was decided to ignore Category 5—the "retired," etc.—which left the following categories of membership:

Category 1 .. .. (the full-time) General Practitioners
Categories 2, 3, and 4 the Other Doctors

All in these four categories who were resident outside the United Kingdom were also excluded. The number of cases in 1947–50 was thus reduced by five, and the size of the sample by about 4%. The small changes in the occupational distribution of the membership from January 1, 1947, to January 1, 1951, were assessed, and the sample of the total relevant membership finally set up was an average for the whole four-year period 1947–50. On the main issue, however, it was clear that some quite independent check of the occupational allotment was necessary. We therefore decided to classify sufficient of the two samples and sets of cases again, and on this occasion from the official National Health Service lists. Only such a procedure, it was felt, would adequately test the categorization already made from the *Medical Directory*. It can be said straight away that the result of this check showed a surprising degree of conformity between the findings from the National Health Service lists in mid-1951 and the allocations already made from the *Directory*; surprising in view of the changes introduced by the N.H.S., the fact that a certain proportion of doctors did not join it, and because a number of men must have retired or died between the date of the *Medical Directory* used for categorization and that of the lists used for checking (see Appendix 3).

The final analysis can now proceed with some confidence. The two 10% samples were pooled, giving a one-in-five sample; as were also the first attacks of 1947–50. In addition, further to increase the numbers, the first attacks at ages 40–44 in the years 1947–50 were now included, and a one-in-five sample of the relevant membership in that age group also was drawn and categorized, making now a grand total of just over 1,100 names. Tables IX, X, and XI assemble the results by age and category, so far as is reasonable with the small numbers involved. Table IX sets out the

TABLE IX.—*Incidence of Coronary Heart Disease in Medical Practitioners—Occupational Comparison, 1947–50. Men Aged 40–64 Inclusive. Average Annual Rates per Thousand*

| Ages Last Birthday (Years) | General Practitioners, Category 1 | Other Doctors, Categories 2+3+4 |
|---|---|---|
| | Incidence | Incidence |
| 40–49 | *4·3* | *1·3* |
| 50–54 | *8·1* | *5·0* |
| 55–59 | *14·4* | *7·2* |
| 60–64 | *18·4* | *11·1* |
| 40–64 incl. | *8·8** | *4·4** |

* Rates standardized for age on the civilian population of England and Wales in mid-1949.

Occupation categories: The classification adopted was intended to distinguish:

Category 1 General Practitioners: Full-time general practitioners.
" 2 Consultants/Specialists: Full-time clinical (including academic) specialists.
" 3 "G.P.-Specialists": Doctors who are both.
" 4 Public Officials: Full-time officials in central and local government; regular serving officers, etc.

*Incidence* is the rate of first clinical attack of coronary heart disease, per 1,000.

For further details, including number of cases involved, see Tables X and XI.

All data are for men in the Medical Sickness, Annuity and Life Assurance Society, Ltd., resident in the United Kingdom, with non-cancellable sickness insurance. (Retired practitioners, etc., have been excluded.)

TABLE X.—*Incidence of Coronary Heart Disease in Medical Practitioners—Occupational Comparison, 1947–50. Men Aged 40–64 Inclusive. Figures in Roman Type=First Clinical Attacks; in Italics=Average Annual Rates per 1,000*

| Ages Last Birthday (Years) | Occupation | | | | | | |
|---|---|---|---|---|---|---|---|
| | (1) General Practitioners. Category 1 | (2) Consultants/ Specialists. Category 2 | (3) "G.P.-Specialists." Category 3 | (4) Public Officials. Category 4 | (5) All Except General Practitioners. Categories 2+3+4 | (6) General Practitioners and "G.P.-Specialists." Categories 1+3 | (7) Consultants/Specialists and Public Officials. Categories 2+4 |
| 40–44 | 9 *3·0* | 4 *1·7* | 1 | 1 | 6 *1·3* | 27 *3·6* | 5 *1·5* |
| 45–49 | 17 *5·5* | | | | | | |
| 50–54 | 19 *8·1* | 7 *4·7* | 7 *7·1* | 3 *6·7* | 9 *5·0* | 24 *8·0* | 4 *3·4* |
| 55–59 | 23 *14·4* | | | | 8 *7·2* | 25 *12·8* | 6 *7·9* |
| 60–64 incl. | 14 *18·4* | 5 *12·5* | 3 *10·7* | 2 | 10 *11·1* | 17 *16·4* | 7 *11·3* |

See notes to Table IX.
*Rates per 1,000* were calculated from the number of first attacks (numerator), and the total numbers corresponding by age and occupation in the Society (denominator). These total numbers were estimated from the sum of two independent 10% samples drawn by single years of age from the relevant membership. No rates are calculated for less than three cases.

TABLE XI.—*Incidence of Coronary Heart Disease in Medical Practitioners—Occupational Comparison, 1947–50. Men Aged 40–64 Inclusive. Average Annual Rates per 1,000—Standardized*

| Ages Last Birthday (Years) | | Occupation | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | (1) General Practitioners. Category 1 | (2) Consultants/ Specialists. Category 2 | (3) "G.P.-Specialists." Category 3 | (4) Public Officials. Category 4 | (5) All Except General Practitioners. Categories 2+3+4 | (6) General Practitioners and "G.P.-Specialists." Categories 1+3 | (7) Consultants/ Specialists and Public Officials. Categories 2+4 |
| 40–59 incl. | No. of first attacks | 68 | 11 | 8 | 4 | 23 | 76 | 15 |
| | Average membership | 2,510 | 960 | 630 | 340 | 1,930 | 3,140 | 1,300 |
| | *Incidence* | *7·1* | *3·1* | *3·2* | *3·9* | *3·3* | *6·3* | *3·3* |
| | Standard error | ±1·0 | ±1·0 | ±1·3 | ±2·1 | ±0·74 | ±0·81 | ±0·88 |
| 40–64 incl. | No. of first attacks | [illegible] | 16 | 11 | 6 | 33 | 93 | 22 |
| | Average membership | 2,700 | 1,060 | 700 | 395 | 2,155 | 3,400 | 1,455 |
| | *Incidence* | *8·8* | *4·5* | *4·3* | *4·7* | *4·4* | *7·8* | *4·4* |
| | Standard error | ±1·2 | ±1·2 | ±1·5 | ±2·1 | ±0·85 | ±0·96 | ±1·0 |

See notes to Tables IX and X.
Average membership estimated from the 20% sample.
The standard errors of the rates quoted above allow for the standardization.
*Standardized rates* are based on the civilian population of England and Wales in mid-1949. (The standardized rates at 45–64 inclusive were Category 1, *10·8* per 1,000; and Categories 2+3+4, *5·5* per 1,000.)

main findings, Table X gives the details, Table XI is a summary table.

Three things stand out:

(1) In this Society, during 1947–50, the full-time general-practitioner members aged 40–64 had about twice as high an incidence of coronary heart disease as the other members (Table IX). The inclusive figures of 8.8 and 4.4 per 1,000 are standardized to take into account the differences of age composition in these two large groups. If Categories 1 and 3, the only ones in which general practitioners are at all likely to be classified, are merged and compared with the remainder (columns 6 and 7 of Tables X and XI), the incidence for this all-inclusive general-practitioner group is still about 1.8 times that of the others.

(2) The excess among general-practitioner members is seen at all ages studied, but is greater at 40–49 years than at 50–64 (Table IX, and columns 1 and 5 of Table X), the ratio of the incidence being:

| | General Practitioners (Category 1) : | Other Doctors (Categories 2+3+4) |
|---|---|---|
| At 40–49 years .. .. | 3·3 : | 1 |
| „ 50–59 „ .. .. | 1·8 : | 1 |
| „ 60–64 „ .. .. | 1·7 : | 1 |

(3) There is, unexpectedly, no indication of an occupational gradient with the intermediate group of "G.P.-Specialists" between the general practitioners and the specialists. Table XI shows that the standardized incidence at 40–59 and 40–64 years of age among general practitioners is different from the rest—the specialists, G.P.-specialists, and public officials; and the composite rate for these three categories as shown in column 5 of Table XI is a fair account of them all (Fig. 11). The numbers, however, in Categories 2, 3, and 4 are very small, so that no conclusions can be drawn about them individually.*

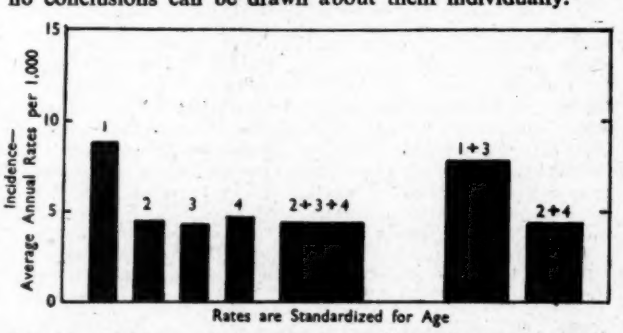


FIG. 11.—Incidence of coronary heart disease in general practitioners and other doctors, 1947–50. Men aged 40–64, inclusive. Category 1, general practitioners; Category 2, consultants/specialists; Category 3, G.P.-specialists; Category 4, public officials. (See Tables IX, X, and XI for further details of membership of Society, etc.)

*Statistical "significance" of the differences in the rates.*—The main difference—that is, that between the rate for Category 1 and the combined rate for Categories 2, 3, and 4, at ages 40–64 inclusive—might appear by chance, if in fact there is *no* occupational difference, less than once in 1,000 times. The significance of the differences between the rate for Category 1 and the rates for Categories 2, 3, and 4, taken separately, is also indicated below:

| Ages | Category 1 and | | | |
|---|---|---|---|---|
| | Category 2 | Category 3 | Category 4 | Categories 2+3+4 |
| $p$ at 40–59 incl. ≏ | 0·002 | 0·003 | 0·04 | 0·001 |
| $p$ „ 40–64 „ ≏ | 0·003 | 0·003 | 0·02 | 0·0007 |

The differences among Categories 2, 3, and 4 are not significant by any standard.

$p$ for the difference between Categories 1+3 and Categories 2+4 is 0·004 both for ages 40–64 and ages 40–59; for the difference between Categories 1+3 and Category 2 alone it is 0·009 for ages 40–64, and 0·006 for ages 40–59.

These values of $p$ allow for the facts that the population was estimated from a sample, and that for each comparison the sign of the difference is consistent in each age group. Consequently, these values of $p$ cannot be deduced directly from the standard errors of the *standardized* rates quoted in Table XI.

The cases are too few to quote many special rates that would be of interest. But it can be said that the findings were rather stable, and the general-practitioner members did have an excess in each of the four years 1947–50; in fatal as well as in non-fatal cases; in angina pectoris as well as in "coronary thrombosis," in diagnoses with and without "specialist confirmation"; and in the different parts of the United Kingdom taken separately. Recalculation of Tables III–VII in terms of general practitioners and other doctors has not yet been undertaken.

## New or Old?

Clearly the next question is, When did this higher incidence of coronary heart disease among the general-practitioner members appear; is it a new or an old phenomenon? Adequate records of the Society for dead members are available only as far back as 1940, and the question therefore becomes, Is the higher incidence among these general practitioners a post-war development, or was the experience of 1940–6 similar to that of 1947–50? Unfortunately, though there are some interesting pointers, it is not possible to answer even this question at all satisfactorily.

To obtain such an answer two sets of facts would be needed about the experience of 1940–6, just as they were needed for 1947–50: (1) the number of first attacks at different ages in each occupational category during 1940–6; and (2) the number of members at different ages in each category during those years. The first attacks of 1940–6 are already known from the study of prognosis, and these can be categorized from the *Medical Directory* of the year preceding the attack. But for various reasons it is quite impracticable to reconstruct the age and occupational structure of the Society in the war years, so that rates comparable with those of Tables IX, X, and XI may be calculated. An idea of the main characteristics of the wartime membership can be obtained, however, if two assumptions may be made: (*a*) if it can be assumed that the occupational distribution of the Society's members in the years 1942–5 was similar to that found *five years later* in 1947–50, in men *five years older*—that is, if it can be assumed that the proportion of general practitioners to other doctors, aged 40–59, in 1942–5 was similar to the proportion observed in the members aged 45–64 in 1947–50; and (*b*) if it can be assumed, further, that the occupational distribution of the members in 1942–5 was reasonably typical of the longer period 1940–6. In defence of these assumptions it may be stated that the membership of the Society is a very stable one, especially over 40 years of age, and it remained particularly stable during the war; the ratio of general practitioners to other doctors, moreover, was found to be similar in each five-year age group during the years 1947–50 except in the oldest age group.

The assumptions are therefore fairly reasonable: nevertheless, with such hypothetical information about the "population," all that can be expected are estimates, rather than facts, about what took place during the war.

In Table XII the 1940–6 material is analysed, so far as the nature of the information permits. The occupational *distribution* of the members in 1940–6 is estimated, as described, by projection backwards; the average *number* of first attacks of coronary heart disease, per year, in men aged 40–59, is given; and the annual averages of cancer deaths at the same ages have been added for comparison. The *ratios* between these average annual numbers are given in parentheses and in italics.

TABLE XII.—*Findings in Men Aged 40–59 Years*

| | 1940–6 | | 1947–50 | |
|---|---|---|---|---|
| | General Practitioners | Other Doctors | General Practitioners | Other Doctors |
| Occupational distribution of membership (Ratio) .. .. .. .. | *1·2* : *1* | | *1·3* : *1* | |
| Average no. of first attacks of coronary heart disease per year .. .. | 6·9 | 6·3 | 17 | 5·8 |
| (Ratio of averages) .. .. | (*1·1* : *1*) | | (*2·9* : *1*) | |
| Average annual number of first attacks of coronary heart disease fatal in the first month .. .. .. | 1·9 | 2·0 | 5·3 | 1·5 |
| (Ratio of averages) .. .. | (*0·9* : *1*) | | (*3·5* : *1*) | |
| Average no. of deaths from malignant disease per year .. .. | 1·6 | 1·7 | 2 | 2·3 |
| (Ratio of averages) .. .. | (*0·9* : 1) | | (*0·9* : *1*) | |

N.B.—There are no *rates* in this table, but merely *numbers* of cases per year in two groups, and *ratios* of these numbers to each other.

There are several points of interest in this table. The *ratio* of the first attacks of coronary heart disease (general practitioners: other doctors) is different in 1940–6 from that in 1947–50 (1.1:1 and 0.9:1, in 1940–6, as against 2.9:1 and 3.5:1 in 1947–50). The ratios in 1940–6, when compared with the ratio of the numbers in the two occupational groups in those years (1.2:1), suggest that there was little, if any, excess coronary heart disease among general practitioners during the war.† If the experience of the "G.P.-Specialists" is added to that of the general practitioners the findings are again unaffected. These figures are interesting in light of the American observation that there was no material difference in the coronary mortality rates of medical specialists and non-specialists in the years 1938–42 (Dublin and Spiegelman, 1948).

Table XII also suggests that the excessive number of first attacks of coronary heart disease among the general-practitioner members in 1947–50 was due more to an increase of first attacks in them than to a reduction in the "other doctors." The relevant figures, as given in the table, are 6.9 and 17 (per annum) for general practitioners, compared with averages of 6.3 and 5.8 for the other doctors. The occupational changes in the membership during 1947–50 are known to be very small, and there is nothing in this, or in the increase even of the *total* number of members with non-cancellable insurance between 1940–6 and 1947–50, or in the ageing of the members within the range 40–59 years, that could possibly account for this more than doubling of the number of first attacks of coronary heart disease among the general practitioners.

Furthermore, Table XII suggests that this increase of first attacks among general practitioners in 1947–50 may represent a real increase, and is not merely the result of improving *diagnosis* with readier recognition of milder, or atypical cases. The average numbers of fatal and non-fatal first attacks among the general practitioners have increased in step. The proportion, therefore, of first attacks which were soon fatal—a proportion that may be used as a crude index of the severity of the cases being recognized—did not change materially in the two periods (1.9/6.9, 27%, almost equals 5.3/17, 31%).‡ The similarity of this fraction in 1940–6 and 1947–50 suggests that there was little change in the level of diagnosis in the two periods. The similarity of these early case-fatality ratios in the general practitioners and other doctors (in the other doctors the ratios in the two periods were 32% and 26%) supports, also, the proposition that the level of diagnosis in these two groups was similar.§

Finally, it may be remarked that the stability of the cancer mortality figures suggests that there has been no general breakdown in the Society's recording system, or any major "experimental error" affecting in particular one section of the membership. This stability, moreover, strengthens the previous impressions of the stability of the membership of the Society.

Such confirmatory arguments, however, cannot substitute for the absence of needed facts. An attempt had to be made to deal with the problem, and this is the best we have been able to do. It will be remembered (Fig. 12; Josiah Macy Jr. Foundation, 1951) that there is some evidence of a diminution of coronary heart disease during the recent war in several European countries, and of a rise after the war. Possibly there was some general factor that operated to produce an "abnormal" occupational distribution among the membership during the war. At the most it may here be said that there seems to have been little, if any, excess of coronary heart disease among general-practitioner members during the war. It appears that there may have been some increase of the disease among these general practitioners since the war, and this increase may be related to the excess of the disease found among them in 1947–50. Because of the hypothetical nature of the 1940–6 "population" it is impossible to say by how much the true average *rate* of first attack among the general practitioners increased from 1940–6 to 1947–50, whether one age group was affected more than another (though the indications are that men in their fifties were affected at least as much as men in their forties), or when the increase began. The higher incidence among these general practitioners, it may be recalled, was present throughout the four years 1947–50. There is unfortunately no other evidence to appeal to for confirmation—or otherwise, and it is a particular pity that analysis of the pre-war situation is impossible.

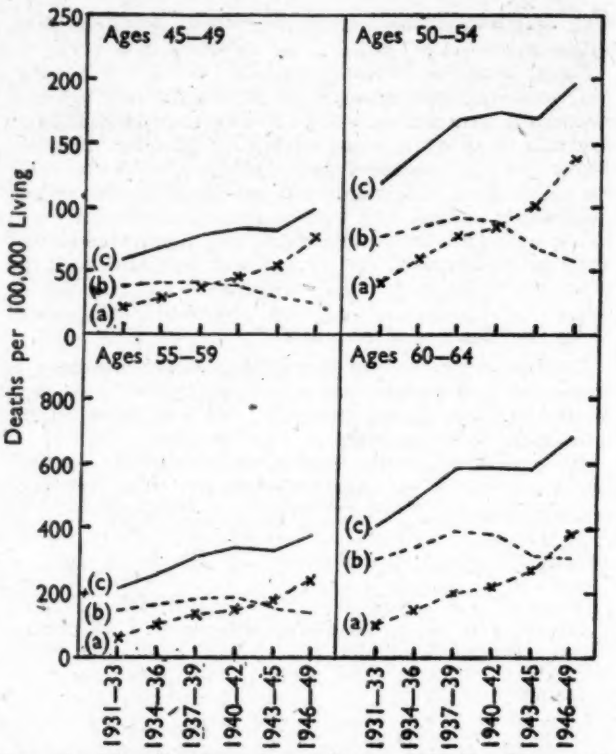


FIG. 12.—Mortality from heart disease, 1931–49. Men aged 45–64, inclusive. England and Wales. (Registrar-General.) (*a*) ×--×--× Diseases of Coronary Arteries, Angina Pectoris. (Int. List No. 94) (*b*) ------ Chronic Myocardial Disease, excluding those specified as Rheumatic, but including Myocardial Infarction. (Int. List Nos. 93 *c*, *d*; 162 *a*, *c*.) (*c*) ——— The sum of (*a*) and (*b*).

Part II, above, is an example of simple epidemiology (Gordon, 1950)—the exploration of rates of incidence in different social groups. This step may of itself make an immediate contribution to aetiology, but its main value is usually to raise questions rather than to answer them, and at the same time to provide an economical "way in" to further study, through comparison, for instance, of the circumstances of high and low incidence. In epidemiological inquiries large-scale hypotheses can often be formulated and tested, supplementary to those of the bedside and laboratory; and hypotheses framed in clinical and laboratory study can often be independently validated. Finally, as an experimental science, epidemiology can try to apply its notions "in the field"—for example, by correcting faults that appear to be relevant and observing the subsequent incidence.

If the situation in this Society is at all representative, interesting and important questions must arise about the health of doctors, and about the possible contribution which doctors might be able to make, through their own health and sickness, to the better understanding of coronary heart disease, and, eventually, to the development of a rational preventive programme. But *is* the experience of the Society true of the whole profession? We are quite unable to answer this question. A great deal of effort could be spent in an attempt to estimate how typical the members of the Society are of all the doctors in the country. For instance,

†The same indication arises if the men *first* attacked by coronary heart disease in 1940–6, who had a *second* absence for this in 1947–50, are categorized. There were 26 such men, aged 40–59, in the four years 1947–50—13 general practitioners and 13 other doctors; that is, an average of 3.3 per year in both groups, and a ratio of 1.0:1. These *further* absences, it should be noticed, occurred in exactly the same population, and in the same period, that showed occupational group averages of 17 and 5.8 for *first* attacks, with a ratio of 2.9:1 (see Table XII).

‡There were, in fact, as already mentioned, some signs of an improvement in this early case-fatality ratio during 1949–50, but this is too slight to affect the argument, the more particularly as better treatment may have contributed to the reduction in fatality.

§Another indication of the level of diagnosis is the number of such less definite diagnoses as "myocardial degeneration," "myocarditis," etc., compared with the definite diagnoses alone accepted as coronary heart disease for the purposes of this investigation. These less definite diagnoses were few in either period, or in either group. The average annual number of "first attacks" of "myocardial" disease in 1940–6 was 1.1 both for general practitioners and other doctors; while in 1947–50 the figures were 1.5 and 1.0, respectively.

a superficial examination of the available statistics indicates that rather more than a quarter of all general practitioners aged 40–64 years appear to be members included in this inquiry, rather less than a quarter of the consultant/specialists. We do not, however, consider this to be the correct kind of approach to the problem. The first thing that should be done is to estimate the incidence of the disease *directly, in the profession as a whole*. Only this could prove how typical are the occupational differences—and apparent similarities—demonstrated here among the *members of this Society*. And only in this way could the relevant associations and relationships of the disease be adequately investigated.

An appropriate field survey would be necessary, and this could conveniently do more than determine the incidence rates in different sections of the profession. Questions could be designed to explore such differences of incidence as may be discovered, in terms of current thinking on the pathogenesis of coronary heart disease, and in terms of the structure of the profession and the different roles within it. To be most fruitful, of course, such a survey should also be based on *ad hoc* case study—for example, of the work, habits, and social relations of general practitioners, of different ages, in different types of practice, recently recovered from "coronary thrombosis," and of "controls." The opportunity should be taken to disentangle the antecedents of coronary heart disease among doctors, whether or no it is confirmed to be commoner in one group of them; it is important enough in all.

Various methods of inquiry are open. It may not be practicable (though it need not be entirely ruled out) to compare the prevalence of the chronic atheroma and the acute thrombosis in different sections of the profession, a study that would be particularly valuable for the understanding of the fundamental processes of coronary heart disease (Morris, 1951). Nor would doctors make easy subjects for investigations of the psychological components of the disease; though such matters as hours of sleep, load of work and responsibility, and the response to upsetting situations are "psychological" as well as "physical." (It is remarkable how little study has been reported on the nature, predispositions, and consequences of medical practice, and on such matters as the emotional satisfactions and stresses involved in different types of professional life.) The distribution of the disease in town and country, and different types of town and different parts of the country, should be examined. The influence of "constitution," of family history in different sections of the profession, and of physique (Gertler, Garn, and White, 1951) could be investigated, together with their possible relation to the "environmental" factors. Considerably more clinical detail than is presented here can be collected. Records of the war history (both wars), as well as post-war experiences of patients and "controls," are needed. Studies of the blood lipids, at present so promising, though where they are leading is still clouded in mystery, may be possible, with a long-term follow-up; and assessments of diet and nutrition. The association and dissociation of other disorders and coronary heart disease might be investigated (see Gregg, 1950). Analogous studies might be carried out on the incidence among doctors of such conditions as "essential" hypertension, duodenal ulcer, and of course cancer. In brief, the occasion might be taken for the profession to examine itself, medically and socially.

Finally, before leaving this discussion of coronary heart disease in medical practitioners, it may be worth pointing out that this study has been made with simple methods and in a small—though well-defined—"population." The number of men studied, in fact, might be found in a town of 30,000 or 35,000 people, a town the size of Hereford, or Taunton, or Airdrie. In view of the growing interest of the medical officer of health in epidemiological inquiries on the non-infectious diseases, it is reassuring to see that they may well be usefully carried out with modest resources, as is already well known to be possible in the acute infections (Paul, 1950). The present inquiry, by its reference to a well-defined "population," by its study of a "cohort," and by its use also of some clinical approaches, has yielded elementary information on the incidence and prevalence of one of the most serious diseases in the population. It has raised questions on the natural history in middle age, and has provided some facts on the life of its victims. It has given some indication of what would be involved in a field survey of the principal cardiovascular diseases, and has suggested one approach to the investigation of causes, through its uncovering of what may be an "experiment of opportunity."

## PART III: THE DISEASE IN MEDICAL PRACTITIONERS AND IN OTHERS

There is a widespread impression that doctors suffer more from coronary heart disease than the rest of the population, though the papers on the subject have been divided (Osler, 1910; Boas and Boas, 1949). This impression is based on clinical experience and mortality rates from England and Wales and the United States (Registrar-General, 1938; Dublin and Spiegelman, 1947). The clinical evidence is of doubtful value. Quite simply, in order to determine whether there is any difference in the experience of doctors and others, it is necessary to know, for a definite period of time, the ratio

$$\frac{\text{No. of cases of coronary heart disease in doctors}}{\text{No. of doctors among whom these cases are occurring}}$$

and it must be possible to compare this with the corresponding ratio

$$\frac{\text{No. of cases in other people}}{\text{population in which these are occurring}}$$

In both it is necessary to know the sex and ages of those in the numerator and denominator. (The problem is exactly the same as in comparing the experience of one group of doctors with that of another.) Many of these facts are not usually available in clinical work, and impressions based on the numbers of doctors seen by any individual or in any clinic are apt to be quite inaccurate. Probably the least reliable impressions are those of distinguished physicians who may have a quite disproportionate number of doctors in their personal practice, and generalize from that. The mortality rates present other kinds of problem: in particular, the latest from this country are for 1930–2, as the war prevented the holding of the 1941 census and the publication of the traditional Decennial Supplement on Occupational Mortality based upon it. The clinical recognition of "coronary thrombosis" was not yet very common in this country in 1930–2. Moreover, as indicated in Part II of this paper, it may not be sufficient to group all medical practitioners together as was done in those death rates.

Since the present inquiry forms part of a wider investigation which includes a number of non-medical groups, it provides an opportunity for reconsideration of this question which has caught the imagination of physicians on both sides of the Atlantic. The non-medical groups include professional, scientific, technical, executive, and clerical grades of the Civil Service (established men only), a large number of schoolmasters, several grades of Post Office workers, the Metropolitan Police, building workers of the London County Council, drivers, conductors, and several other grades of the staff of London Transport Executive, and various smaller professional, clerical, and industrial groups. In all, the diagnoses made among a miscellaneous group of about 200,000 men for 1949 and/or 1950 can be considered here—that is, a number that might be found in a city of a million people or so. Between 35 and 40 years of age there are relatively few cases, and after 60 the conditions of service in the various organizations and grades differ so considerably that it is not always practicable to define who remains and why. The experience of men aged 40–59 years will therefore be considered and, as before, first attacks of the disease will provide the main material for comparison. The information was specially obtained for the purposes of

this research by the various occupational health services in their examinations of workers (especially those absent from work for any length of time), as well as from the certificates given by general practitioners, hospitals, and consultants and sent to employers. A particular check was kept so far as possible on the "suspect labels" of "myocardial" disease, etc. The main findings are presented below.

### Incidence of Coronary Heart Disease

*Standardized Rate per 1,000 Men Aged 40–59*

| | |
|---|---|
| General practitioners .. .. .. | 7.1 (Table XI) |
| Other doctors .. .. .. .. | 3.3 " |
| Miscellaneous, non-medical group .. | 2.5 |

Comparing, first, the experience of the "*other doctors*" and the non-medical group, it may be said that an incidence of 3.3 is little higher than 2.5. If, therefore, the experience of this non-medical group is at all typical, it would appear that doctors in this country are diagnosing first attacks of coronary heart disease about as often in the population as a whole as they are diagnosing it in those members who are not full-time general practitioners.

Here we must digress to emphasize what such figures mean. When an attempt is made to compare the incidence of a disease in one group with that in another, what is in fact being compared is the incidence of the *diagnosis* of the disease in the two groups. Now there is no justification whatever for supposing that the *level* of diagnosis of coronary heart disease is the same in the medical profession as in the public, and therefore no justification for assuming that because similar rates are here reported the true incidence is similar. These medical rates almost certainly include a high proportion of all the coronary heart disease that produced symptoms among the "other doctors." In the miscellaneous non-medical group it is not possible to say more than that the methods of diagnosis and collection adopted *may* have resulted in an equally high proportion of coronary heart disease being recognized and recorded; but quite likely it did not.* If many of the cases in the non-medical group are being wrongly called coronary heart disease, there may in fact be some real excess of it among "other doctors." On the other hand, if many cases of coronary heart disease among the miscellaneous group are being overlooked, it may well be that these "other doctors" have a *lower* incidence than the population as a whole, in so far as this is represented by the group being studied.

The fatal first attack rates show similar trends, and so do the total mortality rates (Fig. 13). In the following, the rates for England and Wales are estimated from the provisional figures for 1950, kindly supplied to us by the Registrar-General (1952, personal communication), and are based on the Sixth Revision of the International List of the Causes of Death. (One effect of this revision is that deaths from myocardial infarction are now included with coronary disease.) These national figures are used because the *total* mortality is required, and there is no means of assessing how many of the deaths from coronary heart disease that do not occur in the first clinical attack will be recorded by employers in the above non-medical group, since the first (non-fatal) attack may result in a change of employment. The rates of medical practitioners are for the Society's members in 1947–50, and they include deaths in later as well as in first attacks.

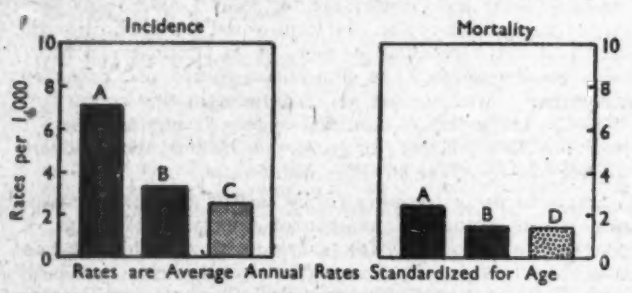


FIG. 13.—Coronary heart disease in medical practitioners and in others. Men aged 40–59, inclusive. A=General practitioners (Category 1), 1947–50. B=Other doctors (Categories 2+3+4), 1947–50. C=Miscellaneous non-medical group, 1949–50. D=England and Wales, Civilian Population, 1950 (provisional). A and B held Medical Sickness, Annuity and Life Assurance Society Immediate Benefit Policies.

### Mortality from Coronary Heart Disease

*Standardized Rate per 1,000 Men Aged 40–59*

| | |
|---|---|
| General practitioners .. .. .. | 2.4 |
| Other doctors .. .. .. .. | 1.5 |
| Males, England and Wales .. .. | 1.4 |

It should be remembered, moreover, in comparing the 1.5 and 1.4 that even under 60 years of age there still are quite a number of deaths ascribed to "myocarditis," etc., in the Registrar-General's Tables, which are not included in the 1.4; among the medical practitioners the number so diagnosed is quite negligible.

The problem is very different when comparing the experience, and particularly the incidence, of the *general-practitioner* members with the non-medical group. If the figures presented here for the non-medical group bear any relation to reality, it may well be that these general practitioners have a higher incidence than that group. Moreover, if these miscellaneous professional, clerical, and industrial workers are at all representative of men in their age groups, and if, similarly, the general-practitioner members of this Society are typical of all general practitioners, the higher incidence reported here may be evidence in support of the common impressions on the subject. The differences noted in these total mortality rates are smaller than for the incidence, owing possibly to recent changes in the incidence of first attacks, fatal and non-fatal, among general practitioner members. If this is the reason it may be an illustration how recent variations in *incidence* rates may not appear simultaneously in the corresponding *total mortality* rates, which, in a disease that is often chronic, must show a time-lag and include deaths from illnesses originating over a long period.

Here this problem of a possible difference between medical practitioners and the rest of the population must unsatisfactorily be left. It is an interesting question, but not at present one with a great deal of meaning. Eventually it may be practicable to carry out large-scale diagnostic surveys of coronary heart disease in the population, with standardized methods, and in the course of these it may be possible to answer this particular question.

### Summary and Conclusions

A study was made of clinical coronary heart disease diagnosed among over 6,000 male medical-practitioner members of the Medical Sickness, Annuity and Life Assurance Society, Ltd., aged 35–64, who held "immediate" non-cancellable sickness insurance, providing benefit, on receipt of a medical certificate, for periods of incapacity to work which last seven days or more. The Society also has knowledge of all deaths in its membership, even when these occur after an illness of less than seven days. Apparently about a quarter of the male doctors of the country in the ages studied belong to the Society, and hold this type of insurance. The report deals only with these doctors, and no information is available about the doctors who do not belong to the Society (or about those who do belong but have other kinds of policy). A full medical examination is given to all applicants for membership of the Society.

The average annual *incidence* of first clinical attacks of coronary heart disease ("coronary thrombosis"—acute myocardial infarction—and angina pectoris) in

*The early case-fatality ratio—that is, the proportion of first attacks that were rapidly fatal—is higher among this miscellaneous group than among these "other doctors." But as the advantage of the "other doctors" may partly be due to better *treatment*, as well as to better *diagnosis* with the recognition of milder cases, and the relevant number of "other doctors" variously affected is small, this aspect cannot be satisfactorily discussed.

these medical practitioners during 1947–50 increased with age, to reach 16.6 per 1,000 at 60–64 years.

The coronary *death rate* in 1947–50 similarly rose with age to 7.4 per 1,000 at 60–64 years; and it accounted for about a third of all mortality in these insured medical practitioners in each five-year age group. The deaths were mainly concentrated in the first attack; in 1940-50, inclusive, 56% of all deaths from coronary heart disease among the members aged 40–64 were "*sudden*," occurring in the first six days of the first clinical attack, and another 14% took place in the remainder of the first month. Coronary deaths occurring after the first month of the first attack accounted for only 30% of the total coronary mortality among these medical practitioners in the eleven years 1940–50.

On current experience, the *probability* that the *individual practitioner* member under 45 years of age who has not yet been attacked by it will be attacked by clinical coronary heart disease before he reaches 65 are about one in five, or 20%. The chances that the individual practitioner under 45 years of age will die of it are about one in fourteen, or between 7.1% and 7.5%.

In 1950 the *prevalence* of coronary heart disease among the members aged 60–64 years was about 9%. The 9% was made up of rather more than 4% "active prevalence"—men who were disabled from work for at least a week during the year because of coronary heart disease; and rather less than 5% with "latent" disease—men who had suffered from the disease before 1950, and were alive in 1950 but not incapacitated from work on account of it during that year for as long as a week.

The *prognosis for life in "coronary thrombosis"* was examined from the onset of the first clinical attack. About 30% of all these men aged 40–64 experiencing first attacks died in the first six days of the first attack—the "sudden" deaths; another 8% died in the remainder of the first month, making 38% in all in the first month. Thereafter the picture is quite different, with 2% dying in the remainder of the first year, and an average of about 2% dying annually in the next six years. Men aged 40–49 at the date of onset of the first clinical attack had apparently a rather lower mortality during the few years that can be observed than those aged 50–64. Of the men who survived their first attack of "coronary thrombosis" about half had no further *absence* for coronary heart disease (or its complications) that lasted as much as a week during the next six years. The average *time lost from work* for coronary heart disease during the next six years by men who survived their first attack was about four weeks per man per year. This figure makes no allowance for part-time work, etc.

The outlook for life of men first presenting with *angina pectoris* was very much better than for the others. During the five years from the date of onset about 11% of them died; their prognosis for life was therefore about the same as in men first presenting with "coronary thrombosis" who had *survived* the first month, with its 38% mortality.

The *mode of first presentation* of coronary heart disease in these medical practitioners aged 40–64 was about 24% as "sudden" death; 55% as less severe "coronary thrombosis"; 17% as the relatively benign angina pectoris; and 4% of miscellaneous "coronary insufficiency," etc. The proportion of angina pectoris increased from 12% at 40–44 to 22% at 60–64; the proportion of the intermediate group of less severe "coronary thrombosis" was smaller in the older men; the proportion of "sudden" deaths as first manifestation changed little with age.

The *membership* of the Society holding these immediate sickness insurance policies was sampled and divided into *four occupational categories*: full-time general practitioners, full-time consultants/specialists, "G.P.-Specialists," and public officials. The *incidence* of first attacks of coronary heart disease in 1947–50 was twice as high among the full-time general-practitioner members aged 40–64 as in the rest. The excess incidence was seen in each five-year age group, in first attacks that were soon fatal as well as in those that were not, and in each of the four years 1947–50. When the "G.P.-Specialists" were added to the full-time general practitioners, the incidence in this composite group was 1.8 times the incidence in the remainder. There was little difference between the rates of the full-time specialists, the "G.P.-Specialists," and the public officials, but the numbers in each of these three categories were very small.

Evidence about 1940–6 is incomplete. Such information as is available suggests that there was little, if any, excess among the general-practitioner members during the war. There is no information on the situation before the war.

Finally, the experience of these medical practitioners was compared with that of a large miscellaneous group of men in the civil services, in the professions, and in industry. The incidence of coronary heart disease in this *non-medical* group was well below that of the full-time general practitioners, and resembled that of the other doctors. A comparison with the provisional mortality rates from coronary disease among the males of England and Wales in 1950 gave similar results.

We are obliged to many for helping us in this investigation: to the Directors of the Medical Sickness, Annuity and Life Assurance Society, Ltd. for allowing us free use of their existing records and for setting up the elaborate additional recording apparatus that was necessary; to Mr. E. A. J. Heath, manager and actuary of the Society, and to other members of its staff; to the Registrar-General and the various chief medical officers and their medical and lay colleagues for providing us with the information from which the non-medical rates of Part III were calculated; to the officers of the Accountant General's Department of the Ministry of Health, and in particular Mr. A. A. Greneski and Mr. H. W. Meyer; to the chief medical officers of the Department of Health for Scotland and the Ministry of Health and Local Government, Northern Ireland; to Dr. L. I. Dublin; to Messrs. J. and A. Churchill Ltd. and Mr. B. S. Stanton, Editor of the *Medical Directory*; to Professor A. Bradford Hill, for most valuable criticism and advice on Part II; to Mr. R. D. Clarke for generous help on the whole paper; to colleagues and friends for many suggestions; and to the secretarial staff of the Unit for much practical assistance.

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## APPENDIX 1

### The Individual Practitioner's Chances: Methods of Calculation

To calculate the probabilities shown by Tables III and IV it was necessary to construct two life tables showing the subsequent history of a hypothetical body of 1,000 practitioners coming under observation at exactly age 35 (the lowest age in both Tables III and IV) as follows:

#### Chance of Getting Coronary Heart Disease (Table III)

At each age from age 35 to age 65 the life table shows:

$l_x$, the number at the beginning of the year of age $x$ to $x+1$, not yet clinically attacked by coronary heart disease.

$c_x$, the number (out of $l_x$ at the beginning of the year of age) clinically attacked during the year of age (these must all be first attacks on account of the definition of $l_x$).

$d_x$, the number (out of $l_x$ at the beginning of the year of age) who die during the year of age (except on account of coronary heart disease, in which case they would be already included in $c_x$).

The number of "living," $l_x$, are thus subject to two mutually exclusive causes of reduction—and it will be seen that $l_x - c_x - d_x$ gives $l_{x+1}$, the number living, and not yet clinically attacked by coronary heart disease, at the beginning of the year of age $x+1$ to $x+2$.

Table III shows the probabilities of being attacked for the first time between age $x$ and age $y$ in the form of numbers per 1,000 at age $x$. These probabilities are given by the formula

$$\frac{c_x + c_{x+1} + \ldots + c_{y-1}}{l_x} \times 1{,}000$$

To construct the life table, estimates were needed of (*a*) the rate of first attack in a population of lives not yet attacked, and (*b*) the rate of death from causes other than coronary heart disease in the same population. For the reasons given in the text, the first attack rate was obtained from data for 1949–50. The number of lives attacked in any given year was estimated from the "total prevalence" figures in Table V and subtracted from the total mean population to give the number of lives not yet attacked. The "non-coronary" death rate among lives not yet attacked was assumed to be the same as the "non-coronary" death rate among the whole group investigated, no closer estimate being practicable. This rate was based on data for 1947–50, since no important changes in death rate had taken place in this period and the larger figures thus available gave a smoother progression of rates with age. This "non-coronary" death rate was thus in fact, though not in theoretical basis, the same as the "non-coronary" death rate used in the life table leading to Table IV.

The rates obtained were those for five-year age groups. To obtain rates for individual ages the group rates were plotted and a smooth curve drawn passing through or close to these points so as to estimate the trend in terms of individual ages, the rates for which were read off. The life table was then calculated.

#### Chance of Dying from Coronary Heart Disease (Table IV)

At each age from age 35 to age 65 the life table shows:

$l'_x$, the number living at the beginning of the year of age $x$ to $x+1$, whether already clinically attacked by coronary heart disease or not.

$d'_x$, the number, out of $l'_x$ at the beginning of the year of age, who die from coronary heart disease during the year of age.

$d''_x$, the remainder of the deaths during the year of age.

The number living at the beginning of the year of age $x+1$ to $x+2$, $l_{x+1}$, is thus given by $l'_x - d'_x - d''_x$.

The probabilities shown by Table IV (in the form of numbers per 1,000 at age $x$) of dying from coronary heart disease between age $x$ and age $y$ (whether already clinically attacked at age $x$ or not) are given by the formula

$$\frac{d'_x + d'_{x+1} + \ldots + d'_{y-1}}{l'_x} \times 1{,}000$$

To construct the life table estimates were needed of (*a*) the rate of death from coronary heart disease, and (*b*) the rate of death from all other causes, in both cases in the total population, whether attacked or not. These were obtained from data of 1947–50 as mentioned in the text. The rates obtained were for five-year age groups and rates for individual ages were found by the process described above.

There is a theoretical objection to this life table, and hence to Table IV, in that the body of lives among whom rates were actually observed is subject to the addition of new entrants who have just passed a medical examination satisfactorily and who therefore have not yet been clinically attacked. The practical effect of this objection must, however, be small in this investigation because the number of new entrants above, say, age 40 is extremely small in relation to the existing membership at these ages. Furthermore, the practical effect, if any, of the existence of a small number of recently selected lives is to underestimate slightly the death rates observed, and thus Table IV cannot on this account exaggerate the position.

A further difficulty, which applies to both life tables, is that where more than one force of decrement is involved the process of passing from the observed rates to the numbers in the life table is theoretically more complex than where a life table showing the single decrement of "death from all causes" is concerned. This comp'exity arises from the fact that the forces of decrement considered are operating simultaneously. In the second life table, for example, a death (in $d''_x$) at time $t$ between age $x$ and $x+1$ is dependent on the same life not having died of coronary heart disease between age $x$ and $x+t$. The details of a theoretically correct process depend on the form of the original observed rate, and the considerations involved are fully discussed by Bailey and Haycocks (1946). In the present inquiry the scale of the investigation and the progression and actual size of the rates observed made it possible to apply the original rates for individual ages directly to a radix—that is, the hypothetical body of "lives" whose subsequent history is shown by the life table—of 1,000 at age 35 without affecting the final figures presented. In other circumstances this would not necessarily be possible.

## APPENDIX 2

### Prognosis: Methods of Calculation

As explained in Part I of the text under "Prognosis," all calculations were based on the 243 first absences for coronary heart disease starting in the years 1940–50, inclusive, among men aged 40–64 inclusive. These 243 cases were divided into three groups: 192 which presented in the first absence as "coronary thrombosis," 42 which presented as angina pectoris, and 9 which presented as "coronary insufficiency," etc. (Some of the cases presenting as angina pectoris had further absences where the condition was diagnosed as "coronary thrombosis," but this did not affect the grouping which was determined by the diagnosis in the first absence.) Attention was mainly focused on the 192 cases of "coronary thrombosis," because the number of cases of angina pectoris gave little scope for analysis. The other nine cases were ignored in this context.

Prognosis was considered from three points of view: (1) *Prognosis for life and death*—the chance of dying of coronary heart disease in a given time, measured from the beginning of the first absence from work on account of it. (2) *Prognosis for further absence*—the chance of being absent again because of coronary heart disease in a given time after the end of the first absence. (3) *Prognosis for work*—the average amount of time lost from work because of coronary heart disease, in a given time after the end of the first absence.

All three concepts were explored with the cases presenting as "coronary thrombosis"—the second and third applying, of course, only to the men who could be followed until they returned to work after the first absence. There were 107 such men out of the original 192; 80 died in the first absence, and five could not be followed to the end of it. For angina pectoris it was only possible to attempt prognosis for life and death.

It was not possible to follow all cases for an equal length of time, if only because an arbitrary date had to be chosen at which observations ceased (June 30, 1951), and the cases arose at different times during the 11-year period. The method adopted was to measure time for each case from the beginning of the first absence, or from return to work after the first absence, according to the particular type of prognosis considered. The observation time was broken up into periods of a year (except for the first year in prognosis for life and death, which was divided into different intervals—see below), and the experience in each year was considered for all those cases which were still under observation at the beginning of the year. The proportion surviving, for instance, to the end of the first year was calculated from the first year's data. From the second year's data the proportion of these survivors who reached the end of the second year was calculated. The product of the two proportions gave the proportion of the original number who survived to the end of the second year, and so on. A cumulative product was thus obtained which gave the proportion surviving up to the end of any given number of years from the beginning of the first absence.

The principle was also adapted to calculate the proportion of those returning to work who had a further absence in a given number of years after such retur , using the proportion having further absences in individual years.

It was necessary to consider the experience year by year. An alternative would have been to calculate the proportion dying before the end of a given year by dividing the number of deaths in that time by the total number of persons observed (suitably adjusted for lapses of observation). This would, however, give undue weight to the experience of the first few years, when the numbers under observation are far greater.

In any given year (or shorter period) expressions had to be found which would give good estimates of the proportion dying (and hence the proportion surviving), or the proportion having a second attack, in the year, allowing for the fact that it was not possible to observe everybody for the whole year.

*Reasons for Lapse of Observation.*—The reasons why men could not be observed for a whole year were as follows: (1) Their policies might terminate during the year, on reaching the age of 60 or 65. (2) The end of the observation period (June 30, 1951) might fall within the year. (3) They might die from (*a*) coronary heart disease or (*b*) another condition.

### Prognosis for Life and Death

The percentage dying in the year from coronary heart disease was calculated from the following formula:

$$\frac{\text{No. of persons dying during the year from coronary heart disease} \times 100}{X+Y+Z}$$

Where X = one year each for all those alive and under observation for the whole year.

Y = the sum of the observed parts of a year for those in 1, 2, or 3*b* above.

Z = one year each for those in 3*a* above who would have been followed for the whole year had they lived; and for the rest, the sum of the parts of a year for which they would have been followed had they lived.

The formula had to be slightly modified in the first year, which was divided into three unequal periods—the first six days, the remainder of the first month, and the rest of the year. This decision was adopted because by far the greatest part of the mortality in the first year (and indeed in the whole natural history of the disease under 65 years of age) occurs in the first few weeks—in fact, in the first few days.

In the first six days Y = 0 because no absence occurred within six days of the policy terminating, and, since no absences starting in 1951 were considered, could not occur within six days of June 30, 1951. For the same reason Z is all deaths from coronary heart disease. In this case, then, X + Y + Z is simply all absences starting in the period.

Similarly, for mortality in the rest of the first month, no absences started within a month of termination of the policy and could not occur within a month of June 30, 1951. X + Y + Z here is all survivors of the first six days.

In the rest of the first year it was necessary to convert all periods of observation, which had been calculated in decimals of a year, to decimals of eleven months.

After the first year there were no special difficulties.

Calculations were made for each 5-year age group from 40–64, age always being reckoned from the start of the first absence.

### Prognosis for Further Absence

This applies, of course, only to those who returned to work after the first absence, and there is no problem of unequal periods in the first year. Numbers were not large enough to attempt the calculation for the men with angina pectoris.

In each year after return to work the percentage of survivors who, not having had a further absence, started one in that year was calculated according to the formula:

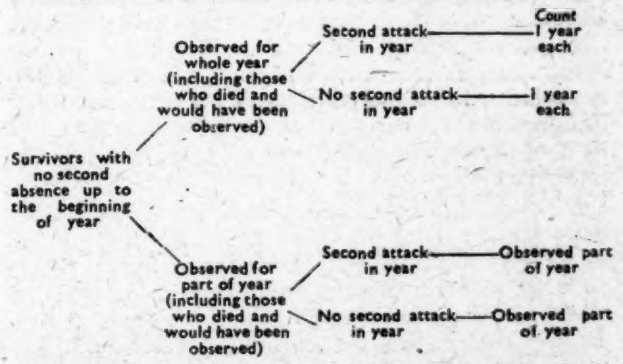
$$\frac{\text{No. of persons starting a second absence in the year} \times 100}{U+V+W}$$

Where U = one year each for all those who, not having had a second absence before the year in question, were alive and under observation for the whole year, *whether or not* they started a second absence during the year.

V = for those who, not having had a second absence before the year in question, were alive but could not be followed for the whole year for reasons 1, 2, or 3*b* above—the sum of the observed parts of a year, *whether or not* they started a second absence during the year.

W = for those in 3*a* above who did not have a second absence before the year in question, and who died during the year from coronary heart disease—one year each for those who would have been followed to the end of the year had they lived and, for the rest, the sum of those parts of the year for which they would have been followed had they lived.

Schematically the matter may be presented thus:


Count
Observed for whole year (including those who died and would have been observed)
Second attack in year
1 year each
No second attack in year
1 year each
Survivors with no second absence up to the beginning of year
Observed for part of year (including those who died and would have been observed)
Second attack in year
Observed part of year
No second attack in year
Observed part of year

"Further absence" includes all cases in which death occurred after return to work from the first absence, even when the fatal illness lasted less than seven days, and was therefore not recorded as an absence.

### Prognosis for Work

As for "Prognosis for Further Absence," this applies only to those who returned to work after the first absence and to the "coronary thrombosis" group.

In each year after return to work the percentage of time lost from coronary heart disease was calculated according to the following formula:

$$\frac{\text{Total time lost from work on account of coronary heart disease in the year}}{\text{Total possible time at work in the year}} \times 100$$

where the "total time lost" is the sum of all periods of absence during the year from coronary heart disease among those under observation at the beginning of the year (including the remainder of the year for those who died during the year); and "possible time" is what would have been possible if there had been no absence—that is X+Y+Z of "Prognosis for Life and Death" if calculated from return to work.

"Time lost" does not refer to the actual working hours. The data merely give the days lost, and these are converted to fractions of a year.

Throughout these calculations exact dates at which absence began and ended, and policies terminated, permitted accurate calculations of the periods of exposure to risk and time off work. The methods would of course not be applicable without adjustment where such complete information was not available. All fractional periods were worked in decimals of a year correct to two decimal places.

The fact that in "coronary thrombosis" a very large proportion of deaths occur in the first few days of the first absence means that what at first seemed a considerable body of data did not give as much information about prognosis for life and death as had been hoped. The information about how prognosis varied with age, for instance, was disappointing. Thirty per cent. of all first attacks result in death within six days and 38% within a month, but, of the 91 *recorded* deaths amongst the 192 cases presenting as "coronary thrombosis," 74, or about 80%, occurred within the first month.

A disease, like pulmonary tuberculosis, in which the chance of early death is relatively small, and in which the deaths are more evenly spaced in time, would give greater scope for the methods used.

## APPENDIX 3

### Independent Categorization of Samples of Membership, and of Men having First Attacks of Coronary Heart Disease, from the Records of the National Health Service

The following are the results of the two checks that were applied to test the validity of the categorizations already made (and summarized in Table XI) from the *Medical Directory* of 1946, 1948, and 1949.

| Category from *Medical Directory* (1) | General Practitioners | | Hospital Staff | |
|---|---|---|---|---|
| | No. Checked on N.H.S. Lists for General Medical Services (2) | Proportion Found on N.H.S. Lists (3) | No. Checked on N.H.S. Lists for Hospital Service (4) | Proportion Found on N.H.S. Lists (5) |
| General practitioners, Category 1 | 485 | 91% | 122 | 1·6% |
| Consultants/specialists, Category 2 | 186 | 1·6% | 87 | 85% |
| G.P.-Specialists, Category 3 | 123 | 88% | 123 | 66% |
| Public officials, Category 4 .. | 68 | 13% | 68 | 2·9% |

Column 1—The four main categories as described in the opening of Part II of this report.

Column 2—All in the samples aged 45–64, and all cases of first attack alive and at work after July 5, 1948, were checked on the general medical services lists of N.H.S.

Column 3—The proportions of those in column 2 who were found to be providing general medical services under the N.H.S. Act. Most of the public officials who were providing such service were clearly doing so for other members of their own staffs.

Column 4—It was impracticable to classify hospital service as "specialist" or otherwise. The following general practitioners are included in the 122 categorized from the *Medical Directory* as general practitioners who were now checked for hospital service: all in the samples who were not found in the general medical services lists; all in the samples who were resident in Scotland and Northern Ireland; all G.P.s who had first attacks of coronary heart disease in 1947–50 and who were alive and at work after July 5, 1948. The 87 consultants/specialists whose names were now checked included all so categorized who were *not* on the staff of teaching hospitals; and all the relevant cases of coronary heart disease.

Column 5—The proportions of those in column 4 now found to be rendering N.H.S. hospital service. The 2 of the 122 categorized as general practitioners who were found to be under contract for hospital service were also rendering general medical services. 12 of the 41 "G.P.-Specialists" not found in the N.H.S. lists seemed to have lost their hospital appointments very recently, judging from the changes in the *Medical Directory* between 1946–9 and 1951; 27 still record these appointments in the 1951 *Medical Directory*; 2 had retired. Two "public officials" now found to be clinical specialists were also clearly continuing as public health officers: 2 of 68 is 2·9%.

# HERPES ZOSTER VARICELLOSUS

BY

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In 1892 von Bókay, of Budapest, described the simultaneous appearance of herpes zoster and varicella in the same patient, and he advanced the theory that the two conditions were caused by the same virus. Seiler (1949) has estimated that the yearly incidence of herpes zoster in Edinburgh is 2 per 1,000 of the population, and that 3.8% of these zoster cases are complicated by a varicelliform eruption.

Most uncomplicated zoster cases in this region are dealt with by local practitioners, and few are referred to the Infirmary. During the period under review (1947–50 inclusive) the number of zoster cases seen was 79, of which 67 were uncomplicated, 6 suffered from involvement of the eye, and the remaining 6 cases were complicated by a generalized varicelliform eruption. Five of the cases are summarized in the Table on the opposite page, and the sixth is described in greater detail below.

### Case Reports

*Case 6.*—A man aged 69 was suffering from haemorrhagic herpes zoster in the distribution of the ophthalmic division of the fifth cranial nerve on the right side. The condition had been present for seven days. Before the attack of zoster his health had been excellent and there was no history of his having taken drugs of any sort. Blood examination showed no abnormality. He had never had herpes zoster or varicella previously. The day after the appearance of the zoster lesions a generalized varicelliform eruption developed suddenly. This eruption reached its maximum distribution within 24 hours, and thereafter no fresh lesions developed. He was photographed on the day he first reported, and a biopsy of a vesico-pustule on the right forearm was done by one of the doctors in the department.

*Case 7.*—The doctor who had done the biopsy on Case 6 complained of general malaise, anorexia, and headache 13 days later, and on the following day a typical varicella eruption developed, beginning in the scalp and on the fauces, and appearing in crops on the trunk, back, face, and arms during the subsequent three days. At its height two days later the rash was profuse, especially on the back, but by this time the constitutional symptoms had subsided. There was no previous history of herpes zoster or of varicella, although the patient had often been in contact with both diseases. He was clear of varicella lesions in 16 days.

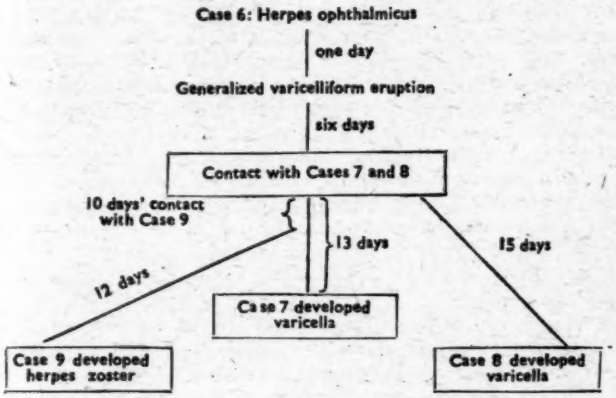
*Case 8.*—The technician who had photographed Case 6 complained of general malaise and had the typical features of varicella 15 days after his one and only contact with Case 6—that is, two days after Case 7 had developed varicella.

FIG. 1.—Case 3, showing haemorrhagic herpes zoster of L1 and L2 and the varicelliform eruption of trunk and limbs.

The technician did not touch Case 6 nor did he handle his clothing. Here again there was no previous history of herpes zoster or of varicella. The rash cleared in three weeks.

*Case 9.*—A doctor, with whom Case 7 had lunched on several occasions between the first and tenth days of the incubation period of his varicella, developed severe neuralgic pains in the right lumbo-sacral region two days after Case 7 developed varicella—that is, 15 days after Case 7 had been in contact with Case 6. Seven days later an eruption of herpes zoster appeared over the area of distribution of the twelfth thoracic and first lumbar nerves on the right side. There was no actual contact by touch between Cases 7 and 9. The doctor in question had often been in contact with cases of herpes zoster and of varicella in the past without contracting either disease, but had not recently been in contact with any other known possible source of infection.

The Diagram illustrates the relationship between the primary zoster and the secondary varicelliform eruption in Case 6 and the spread of infection to three other individuals.




*Summary of Five Cases of Concurrent Herpes Zoster and Varicella*

| | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 |
|---|---|---|---|---|---|
| Sex and age .. | Male; 67 | Male; 82 | Male; 75 | Male; 64 | Male; 79 |
| Distribution of zoster | C3, 4, and 5 on left side | Trigeminal nerve on left side | L1 and L2 on left side (Fig. 1) | 7th nerve rt. side. Ramsay Hunt syndrome | C3 on left side |
| Interval between development of zoster and varicella | 2 days | 1 day | 2 days | 1 day | Less than 4 days |
| Previous history of zoster or varicella | Nil | Nil | Nil | Nil | Nil |
| History of contact with cases of zoster or varicella | " | " | " | " | 15 days' contact with a convalescent zoster case |
| General health | Suffered from chronic lymphatic leukaemia | Diabetic patient | Chronic lymphatic leukaemia treated with *x* rays | Good | Very good |
| Duration of zoster eruption | 6 weeks | 15 days | 7 weeks | Residual facial palsy 7 months later | 15 days |
| Duration of varicella eruption | 14 days | 12 " | 18 days | 15 days | 8 " |

## Laboratory Investigation

*Histology.*—Biopsies were taken from the edge of the zoster eruption and from a secondary vesicle on the trunk in Case 3 and from a secondary vesicle on the forearm in Case 6 (Figs. 2 and 3). The histological features in all

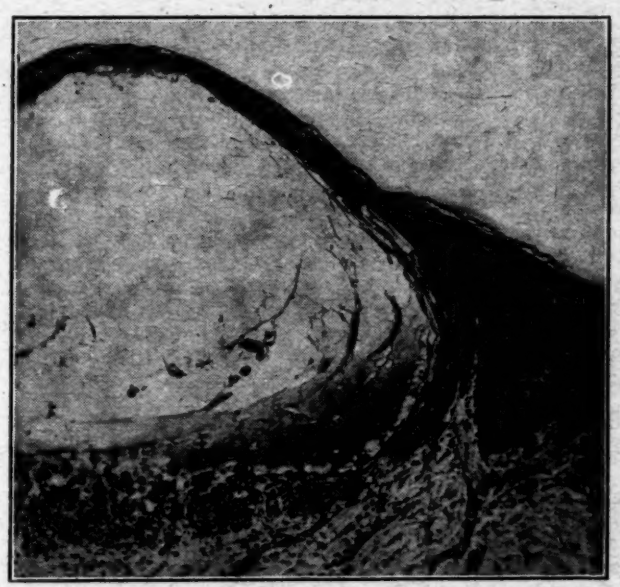


FIG. 2.—Case 6. Histopathology of a varicelliform lesion from the anterior aspect of the left forearm. The stratum corneum is intact, and immediately subjacent to it and to the stratum granulosum a thin layer of degenerated rete cells is visible. The basal layer has disintegrated, with the result that balloon cells, giant cells, and cellular debris lie directly on the upper dermis. H. and E. (× 60.)

FIG. 3.—Case 6. Histopathology of a varicelliform lesion from the anterior aspect of the left forearm. The cells forming the floor of the bulla show varying degrees of degeneration. Intracellular oedema gives rise to the appearance of ballooning of the cells (A); several so-called giant cells (B), consisting of aggregations of degenerated nuclei surrounded by a variable quantity of cytoplasm, are prominent. The infiltrate consists of polymorphs and, to a less extent, of lymphocytes. H. & E. (×450.)

three sections show an intra-epidermal bulla, the roof of which is formed by stratum corneum, stratum granulosum, and the upper part of the degenerated rete mucosum. It is clearly demarcated laterally from the adjacent epidermis by a few rows of flattened degenerated rete cells. The base is composed of degenerated and disorganized rete and basal cells, around which there is an infiltration of polymorph leucocytes and lymphocytes. In this area there is intercellular and intracellular oedema, giving rise to the appearance of ballooning of the cells, and several so-called "giant cells," consisting of aggregations of degenerated nuclei surrounded by the remains of the cytoplasm of these cells, are apparent. The bulla fluid contains some of these balloon cells, giant cells, and leucocytes. In the upper part of the dermis there is oedema, with a well-marked polymorph and lymphocytic infiltrate, particularly in the perivascular situation.

*Bacteriology.*—On admission 10 ml. of blood was withdrawn and fluid was collected from bullae on the groin and thigh in Case 3. Large numbers of elementary bodies were demonstrated in the bulla fluid by the methods of Weiss and of Paschen. The bulla fluid was diluted and set up for complement fixation against three sera derived from known severe cases of herpes zoster, including serum from Case 2, and against three normal sera. The antigens in the vesicle fluid combined with the antibodies in the known positive sera to fix 2 M.H.D. of complement when the sera were diluted 1/16. No fixation took place with the normal sera. No antibodies were demonstrated in the patient's serum, against his own bulla fluid, or against fluid from another case of herpes zoster, indicating that the patient had not at that stage produced antibodies to the herpes zoster virus.

## Comment on Case Reports

In each of the six cases of herpes zoster complicated by a varicelliform eruption the herpes zoster eruption developed first, and the interval between the appearance of the zoster and the development of the secondary rash was 24 hours to four days. None of the patients had previously suffered from herpes zoster or varicella. The herpes zoster and the secondary eruptions both healed with scarring; the duration of the zoster eruption varied within wide limits, from 15 days to 7 weeks, whereas the secondary eruption, which developed to its maximum extent in 24 hours, cleared within 8 to 21 days. In each instance the secondary eruption had cleared before the primary zoster lesions. In Cases 7 and 8 the incubation period of the varicella was 13 and 15 days, respectively—that is, within the normal incubation period of varicella. The incubation period of herpes zoster is said to lie between 9 days (Kundratitz) and 21 days (Siegl); in Case 5 it was 15 days or less, and in Case 9 it lay between 12 and 22 days.

Although epidemics of zoster have been described the actual mode of spread has not yet been established. In 1900 Haslund noted that herpes zoster was often ushered in with nasal or pharyngeal symptoms, and he regarded the tonsils as the probable portal of entry. He thought that the virus was then carried by the blood stream to a posterior root ganglion and thence via a sensory nerve to the skin. Fenner (1948) has described the mode of spread of the virus causing ectromelia in mice. He has proved experimentally that the virus multiplies at the source of infection—normally minor trauma on the skin surface—and spreads thence to infect the associated lymph nodes, where further reproduction results in a blood infection or viraemia. This primary viraemia is controlled by the phagocytic action of the cells of the liver, spleen, and other tissues. When these cells have been overcome by the virus a secondary and more profound viraemia develops, resulting in the clinical condition. It is possible that a similar sequence of events occurs in zoster and varicella, the virus being neurotropic in the former disease and dermotropic in the latter.

Montgomery (1921) considers that the more probable route of entry is via the sensory nerve endings in the skin, and this view is upheld by van Rooyen and Rhodes (1940). It is difficult to conceive how this theory can find acceptance, as prior to the onset of zoster there is rarely any history of trauma over the area affected, and the application of vesicle fluid to the unbroken skin has failed to produce either local or generalized reactions or to stimulate the development of immunity. Furthermore, aberrant vesicles are seen quite often in herpes zoster, and their presence would be difficult to explain on the basis of this theory. Case 7 had been in close contact with Case 6 while doing the biopsy, but infection in Cases 5, 8, and 9, where contact was not close, was in all probability by droplet infection, and the portal of entry may well have been the nasopharynx.

Two cases in this series suffered from chronic lymphatic leukaemia, one of them having been treated with wide-field *x*-ray therapy six months before the zoster attack. A series of 45 such cases developing zoster following irradiation is described by Ellis and Stoll (1949).

## Brief Review of the Literature

The following clinical observations have been advanced in support of the theory that herpes zoster and varicella are caused by the same virus: (*a*) Varicella often occurs in children in contact with cases of herpes zoster. Adults who have not previously had chicken-pox or herpes zoster are also liable to infection. (*b*) Herpes zoster is occasionally followed, within five days of the vesicular phase, by a generalized varicelliform eruption (see Cases 1–6). (*c*) Cases of herpes zoster associated with a varicelliform eruption are not infrequently followed by cases of varicella in contacts, the incubation period being 12–16 days (Cases 7 and 8). (*d*) A few cases have been reported in which varicella has been followed by herpes zoster in contacts (Case 9).

A common origin for the two diseases is supported by the experimental work of Lipschütz and Kundratitz (1925) in Vienna and of Bruusgaard (1932) in Oslo. These authors reported that when infants or young children were vaccinated with zoster vesicle fluid varicelliform lesions developed locally around the site of inoculation; in a few cases a generalized varicelliform eruption made its appearance, and some of the contacts of these cases developed varicella. Cross complement-fixation reactions have been demonstrated between the serum of convalescent herpes zoster cases and the vesicle fluid or crusts of varicella cases, and vice versa, important work in this respect being that of Netter and Urbain (1924) in France and that of R. T. Brain (1933) in this country. Hassko *et al.* (1938) were unable to confirm these findings, and Thomsen (1934) failed to demonstrate any fixation between zoster serum and varicella antigen in 83 cases, the converse reaction with varicella serum and zoster antigen being also negative.

R. T. Brain considers that zoster may be regarded as a neurotropic modification of the dermotropic virus of chicken-pox. He points out that the virus in question most readily attacks young children who have no natural immunity, giving rise to the characteristic eruption of chicken-pox in varying degrees of severity, some of the milder cases passing unnoticed as subclinical infections. In either event the virus is widely disseminated throughout the body and must reach the central nervous system, where it may persist, without causing symptoms, for many years. After a short or long interval the dormant virus is provoked to activity by some autogenous factor or as the result of trauma, a toxic drug, or other infection.

Chicken-pox is a common disease and herpes zoster is relatively common, and those who uphold the dualistic theory contend that their association is not more frequent than might be expected in the case of any two diseases whose aetiology is quite distinct. In favour of this view the following points have been advanced: (*a*) Herpes zoster has often been described in patients who have had varicella

at an earlier date, and it does not therefore appear that a high degree of immunity exists between the two diseases. (*b*) The experiments of Lauda and Stöhr (1926) did not support those of Lipschütz and Kundratitz. They inoculated 55 children with herpes zoster vesicle fluid, but failed to obtain a positive local reaction in a single case. Nevertheless, three of the children developed varicella 14 days after the inoculation. (*c*) Zoster has been described in isolated communities where varicella is unknown, as in Cantor's (1921) report on Christmas Island and Woolley's (1946) report on three cases on Tristan da Cunha. The numbers involved in both these instances were very small, and so the reports should be treated with reserve.

It is still undecided whether the two conditions are due to the same virus, to distinct but antigenically similar viruses, or to a mutation of a reversible nature in the structure of the organism. Under the electron microscope the virus of herpes zoster is indistinguishable from that of varicella; from the clinical and pathological viewpoints this organism has an affinity for skin and nerve tissue. In children and in adults who have not previously had either disease or have not been exposed to subinfection of the virus, the tendency is for an infective dose of the virus to result in varicella, as a result of which a high degree of immunity to the organism is usually established. In the majority of adults repeated exposure to and subinfection by the virus seem to result in partial immunity of the individual. W. Russell Brain (1931) considered that the sensory neurone had a tissue immunity feebler than that of other parts of the body, or that its immunity might be temporarily diminished by a preceding lesion, so that an infective dose of the virus in adult life tended to produce herpes zoster.

## Summary

Six cases of herpes zoster varicellosus are described. In one of these cases two contacts developed varicella within the incubation period of that disease, and during the incubation period one of these two varicella cases was responsible for conveying the infective agent of herpes zoster to another contact. The literature on the subject is briefly reviewed. It is concluded that herpes zoster and varicella are very closely allied; the definition of the exact relationship of the organisms must be left to the bacteriologist.

I wish to thank Professor G. H. Percival for helpful advice in preparing this paper. Thanks are also due to Dr. R. H. A. Siwain for the bacteriological investigations and to Mr. T. C. Dodds for the histological preparations and photographs.

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# PULMONARY ASPERGILLOSIS FOLLOWING POST-INFLUENZAL BRONCHOPNEUMONIA TREATED WITH ANTIBIOTICS

BY

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The association of fungus infections with antibiotic therapy is now well recognized. Although many references are to be found in the American literature, few examples have been recorded in this country. We therefore think the following case of pulmonary aspergillosis is of interest.

## Case Report

A housewife aged 51 was admitted on February 9, 1951, during an influenza epidemic, with a diagnosis of post-influenzal bronchopneumonia and toxic purpura. Her symptoms, which began three weeks previously, were typical of the prevalent influenza. The cough became worse, though the sputum was scanty, mucopurulent, and never blood-stained. She remained febrile, and three days before admission developed a purpuric rash.

*Clinical Examination.*—The patient was slightly cyanosed, dyspnoeic, and febrile—temperature 99.5° F. (37.5° C.), with a dry mouth—and obviously very ill. There were petechiae and haemorrhagic bullae on her face, arms, and legs which were considered to be typical of a "sedormid" eruption. She had taken three tablets before this rash appeared. There was slight impairment of the percussion note at the right base, and diffuse moist sounds were heard over both lower lobes. She had a regular tachycardia of 120, slight venous engorgement, and a little oedema of the ankles suggesting mild congestive failure. The blood pressure was not recorded because of bullae on the arms. A radiograph of the chest on February 9 showed obliteration of the right costophrenic angle with heavy lung shadows and only slight mottled opacities in both lower zones.

*Special Investigations.*—A blood count showed: Hb, 94% (Haldane); white cells, 18,000 (neutrophils 95%, lymphocytes 1.5%, monocytes 3%, metamyelocytes 0.5%); platelets, 230,000 per c.mm.; clotting-time, 5 minutes (capillary tube); bleeding-time, 3¼ minutes (Duke). The urine contained: albumin, a trace; sugar, a trace on occasions; pus cells, 1–4, and red cells, 0–3, per ¼ field. Blister fluid was sterile. Direct examination of the sputum on February 12 showed numerous pus cells and a few yeast forms; no acid-fast bacilli were seen. Culture yielded a heavy growth of *Bact. coli* sensitive to streptomycin and chloramphenicol, and a heavy growth of *Candida albicans*.

*Progress and Treatment.*—Treatment was begun at once with crystalline penicillin, 500,000 units eight-hourly. Two days later the patient was afebrile, the pulse rate had fallen, and she was much improved. On the fifth day, however, despite the absence of fresh physical signs in her chest, the temperature rose, penicillin was discontinued, and chloramphenicol was given, 3 g. initially and 0.5 g. six-hourly. By the tenth day her temperature was 100° F. (37.8° C.); she had a pleuritic pain in the right chest, auricular fibrillation had developed, and the sputum, still scanty, became purulent and streaked with blood.

FIG. 1.—Radiograph of chest (February 19) showing opacities in left upper and mid-zones.

A chest radiograph on the eleventh day (February 19) showed extensive mottled opacities in the left upper and mid-zones containing what appeared to be irregular thin-walled cavities. Streptomycin, 0.5 g. twice daily, was now given in addition to chloramphenicol. On February 20 the sputum contained numerous pus cells, with occasional yeast forms and filaments suggestive of *C. albicans*; no acid-fast bacilli were seen. Culture yielded a predominant growth of *C. albicans*. From that time the patient's condition deteriorated, and she died in coma 16 days after admission.

### Post-mortem Findings

The body was that of a thin elderly woman. There were several bullae on the arms, face, and feet which did not appear infected. Both lungs were emphysematous and oedematous. The left upper lobe contained a thin-walled cavity 5 cm. in diameter which had a trabeculated lining dusted with fine white and grey particles. The rest of this lobe showed bronchopneumonic consolidation, as did the right lower lobe to a lesser extent. The pancreas showed a haemorrhagic area in the tail, with several areas of softening elsewhere. The kidneys were pale and their capsules stripped easily. On the right side there were two large recent infarcts, and on the left several smaller less recent areas of infarction.

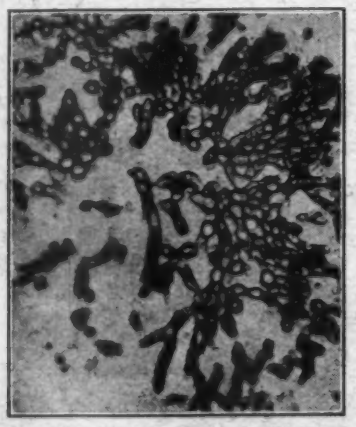

FIG. 2.—Section through lining of cavity in left upper lobe, showing fungal mycelium typical of aspergillus. H. & E. (×320.)

*Histology.*—Sections from the left lung showed the cavity to be lined with a densely felted layer of fungal mycelium and occasional conidia. The structure of the mycelium and conidia was typical of aspergillus (Fig. 2). Neither mycelium nor yeast forms of *C. albicans* were seen in the lung tissue. The adjacent lung showed consolidation, with a haemorrhagic fibrinous exudate. Fragments of mycelium were found in the lung tissue throughout the upper lobe and to a less extent in the lower lobe, where there was a less-pronounced exudate. Sections from the right lung showed some bronchopneumonic consolidation in the lower lobe, with fragments of mycelium in the exudate filling some bronchioles. In the pancreas there were irregular areas of haemorrhage and necrosis. One artery showed necrosis of all coats and scanty cellular infiltration with polymorphs, lymphocytes, plasma cells, and reticulo-endothelial cells, together with surrounding fibrosis. Several blocks from the kidneys showed a number of small arteries with areas of medial fibrosis. No fungal mycelium was seen in the sections from the pancreas or kidney.

*Bacteriology.*—Material from the lung cavity showed conidia typical of the *Aspergillus* genus. Cultures yielded a heavy growth of *Aspergillus fumigatus* Fresenius, together with a scanty growth of *C. albicans*, coagulase-negative staphylococci, and haemolytic streptococci, not of Lancefield groups A, C, or G.

In view of these findings the cause of death was considered to be toxaemia due to post-influenzal bronchopneumonia complicated latterly by aspergillus infection. Neither the purpura nor the presence of *C. albicans* seemed to contribute significantly.

### Discussion

The occurrence of primary aspergillus infection of the lungs is well recognized, particularly in France and America. It occurs most often in patients who are in contact with fungus-contaminated grain, such as agricultural workers and pigeon-feeders. The clinical manifestations of this disease may briefly be said to fall into three groups: the bronchitic, the acute bronchopneumonic, and the chronic granulomatous types, the latter simulating tuberculosis (Virchow, 1856; Renon, 1897; Van Ordstrand, 1940; Cooper, 1946).

Secondary infection associated with other lung diseases is also described, and may occur in tuberculous cavities, bronchiectasis, and even bronchogenic carcinoma (Lapham, 1926; Kampmeier and Black, 1934; Donaldson *et al.*, 1942). Sometimes a blood-borne dissemination gives rise to endocarditis, granulomatous lesions in many organs, meningitis, and even brain abscess (Geiger *et al.*, 1945; Cawley, 1947; Grekin *et al.*, 1950).

The interest of our patient lies in the rapid development of a cavitating bronchopneumonia while massive doses of antibiotics were being administered. The extent of the lung disease shown radiologically was greater than expected from the physical signs, but was in keeping with her progressively deteriorating condition. The necropsy findings confirmed the presence of bronchopneumonic consolidation and of cavitation in the left upper lobe, the wall of which was lined with mycelium. Mycelium was also found in other parts of the lung.

Recent reports have shown the frequent association of fungus infection with antibiotic therapy. Thus Woods *et al.* (1951) describe a series of 25 cases of moniliasis apparently a direct sequel of the use of antibiotics. Of these, 20 had oro-pharyngeal, 3 intestinal, and 2 broncho-pulmonary moniliasis. They suggest that suppression of bacteria usually competing for food with the coexisting *C. albicans* is probably the most important factor in the development of these infections. The disturbance of vitamin synthesis due to alteration of bacterial flora in the gastro-intestinal tract may also be of importance. Harris (1950) reports some success with vitamin-B complex in the prevention and treatment of moniliasis following antibiotic therapy. Foley and Winter (1949) suggest that penicillin may actually enhance the growth and pathogenicity of *Candida* species.

Tomaszewski (1951) recently analysed the side-effects of 126 cases treated with chloramphenicol and "aureomycin"; he remarks on the frequency of a fungal flora in the oral cavity, with a disappearance of the normal bacterial flora. Of particular relevance to our case is the report by Zimmer-

man (1950) of three cases of mycotic endocarditis, two of which were due to aspergillus infection, following antibiotic therapy.

We would suggest that in this case, also, the use of penicillin and chloramphenicol predisposed the damaged lung to infection with *Aspergillus fumigatus*, and that the terminal condition was due to the fungus rather than to any pathogenic bacteria. The case has been presented, therefore, to illustrate one of the dangers of antibiotic therapy—a subject of considerable interest at the present time. With the increasing use of antibiotics, similar cases may be seen more often in the future.


### Summary

A middle-aged woman was treated with penicillin, streptomycin, and chloramphenicol for post-influenzal bronchopneumonia. At post-mortem examination the presence of *Aspergillus fumigatus* infection of the lungs was discovered. The relation of the infection to antibiotic therapy is discussed.


We are indebted to Dr. R. S. Bruce Pearson, Professor H. A. Magnus, and Dr. A. C. Cunliffe for advice in the preparation of this paper; and to Major H. A. Dade, of the Commonwealth Mycological Institute, Kew, for confirming the species of *Aspergillus* isolated.


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# PERIPHERAL NEURITIS IN SYSTEMIC LUPUS ERYTHEMATOSUS


BY

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Peripheral neuritis has not been described as a feature of systemic lupus erythematosus, although it is well known in the closely related condition of polyarteritis nodosa.

The course of systemic lupus erythematosus has been fully described by many authors, notably Libman and Sacks (1924), Keil (1933), Baehr, Klemperer, and Schifrin (1935), Klemperer, Pollack, and Baehr (1941), and Cluxton and Krause (1943), but none of these makes reference to peripheral neuritis, nor do the case reports of Kaposi (1872), Goeckerman (1923), Keefer and Felty (1924), Jarcho (1936), Fergusson, Milne, and Shand (1949), Beare (1949), or Gold (1951). Ginzler and Fox (1940) report bilateral wrist-drop in their patient twenty-four hours before death, but without investigation into its cause, clinical or pathological.

No mention of systemic lupus erythematosus is made by Richards (1951) in a comprehensive review of ischaemic lesions of peripheral nerves.

The following case is therefore put on record.

### Case Report

A housewife aged 34 was originally admitted to the Maida Vale Hospital for Nervous Diseases on June 23, 1949, with a five-weeks history of weakness of the legs and paraesthesiae in her hands and feet. In March she had had a febrile illness, associated with a rash, mainly on the face, that had been diagnosed as rubella. A right-sided pleurisy in 1947 was the only past medical history of note, and she had no recollection of having taken any drugs. There was no significant family history of illness.

Examination revealed a persistent pyrexia of 102 to 103° F. (38.9 to 39.4° C.), with a tachycardia of 100 to 120 a minute associated with marked malaise and wasting. An erythematous maculo-papular eruption was seen on the cheeks, bridge of the nose, angles of the jaw, and wrists. The mucous membranes were generally pale, but there was inflammation of the mouth, tongue, and pharynx. The lymph nodes were not enlarged and neither the liver nor the spleen was palpable. No abnormality was found in the respiratory system, abdomen, or cardiovascular system, and the blood pressure was 135/85 mm. Hg.

In the nervous system, the upper limbs showed a bilateral motor weakness, most marked peripherally, and all the tendon reflexes were absent. Sensation was normal apart from slight loss of two-point discrimination in each hand. A generalized flaccid weakness of the lower limbs was again most noticeable in the periphery, knee-jerk and ankle-jerks were absent, and there was a bilateral flexor plantar response. Light-touch sensation was absent in both legs up to the knees, and vibration sense, while impaired up to the knee on the right side, was completely absent on the left. Pin-prick sensation was unaffected. Calf tenderness was marked in both legs.

The urine showed a heavy cloud of albumin, a large number of erythrocytes, and a few pus cells but no casts. Numerous investigations were negative, including agglutination reactions, the blood W.R. and Kahn test, and an *x*-ray film of the chest. A blood examination revealed: haemoglobin, 73% (Haldane) (10.8 g. per 100 ml.); red cells, 4,300,000 per c.mm.; white cells, 4,420 per c.mm. (neutrophil polymorphs 2,800, eosinophils 360, lymphocytes 1,260 per c.mm.). Lumbar puncture showed a pressure of 170 mm., with no block, 120 mg. of protein and 695 mg. of chlorides per 100 ml., and 5 lymphocytes per c.mm.

Owing to the development of occasional pulmonary crepitations, a course of penicillin and "sulphatriad" was given, but there was no clinical improvement. Vitamin B was administered by injection and later by mouth with nicotinamide and ascorbic acid, but, beyond relieving the stomatitis to some extent, did not appear to have any effect.

Blood culture was negative on admission, but on three subsequent occasions *Bact. faecalis alkaligenes* was grown, sensitive to streptomycin *in vitro*. A week's treatment with this antibiotic (0.5 g. six-hourly) and a further course in August brought no improvement.

A slight generalized lymph-node enlargement developed at this time, but the spleen remained impalpable throughout. Lymph-node biopsy from the left axilla and a sternal marrow examination by direct smear showed no significant abnormality.

Anaemia and leucopenia developed slowly, and by July 27 the haemoglobin was 45% (Haldane) (6.6 g. per 100 ml.), and there were 2,400 white cells per c.mm. (neutrophil polymorphs 1,600, lymphocytes 600, monocytes 200 per c.mm.). On this day 3 pints (1.7 litres) of Group A Rh-negative blood was given, raising the haemoglobin to 70% and improving the general condition, but not reducing the temperature.

By now some improvement in the peripheral neuritis was apparent, although the tendon reflexes were still absent and

the wasting was no less obvious. A further blood transfusion of 3 pints (1.7 litres) was given on August 19, when the haemoglobin had fallen to 60%.

On the following day five epileptiform seizures occurred with loss of consciousness and movements of the head and eyes to the left. For the remainder of the illness bilateral extensor plantar responses were present and bilateral retinal exudates were also noted. Lumbar puncture on August 22 showed 90 mg. of protein per 100 ml. and 12 lymphocytes per c.mm.

The skin lesions had gradually disappeared and by August 25 there was only a slight scaling in the affected areas. The blood pressure now was 120/90 and the urine showed a heavy precipitate of albumin. The heart was not enlarged and there were no significant murmurs and no pericardial rub. Her general condition remained grave, with a pyrexia of 100 to 102° F. (37.8 to 38.9° C.) and a tachycardia of 110 to 130 a minute. On August 25 she was transferred to St. Mary's Hospital.

The relevant investigations thereafter included: Plasma proteins 4.8 g. per 100 ml. (albumin 2.1 g., globulin 2.7 g.); E.C.G., low voltage in all leads with flat or absent T waves. Muscle biopsy showed no evidence of polyarteritis nodosa.

A course of chloramphenicol was given (0.25 g. six-hourly for six days), and a further course of penicillin (500,000 units twice daily), but with no effect. Late in November the temperature fell to normal, but the tachycardia persisted and her general condition deteriorated. Oedema developed, with a large ascites and a right pleural effusion, and the blood pressure rose steadily to 170/120. Severe abdominal pain occurred on December 16, and on the 18th there was evidence of acute generalized peritonitis. A laparotomy was undertaken, but the patient died on December 18 while on the operating table.

### Necropsy Report

Necropsy was performed on December 19. *Cardiovascular system:* The visceral pericardium had a patchy gelatinous appearance and the myocardium was pale brown in colour with scattered paler areas throughout its substance. The endocardium, valves, and coronary arteries appeared normal. The heart weighed 375 g. *Respiratory system:* There was a small right hydrothorax and both lungs were very oedematous. *Digestive system:* The peritoneal cavity contained a large amount of turbid fluid. *Haemopoietic system:* The spleen was of normal size, firm, and congested, and had a small infarct. The Malpighian bodies and trabeculae were prominent. *Genito-urinary system:* The kidneys, each weighing 220 g., were pale in colour, and prominent glomeruli and small scattered haemorrhages were visible in the cortex, which was of normal width. *Nervous system:* No macroscopic changes were noted.

### Microscopical Examination

*Cardiovascular System.*—The pericardium was thickened and showed a diffuse cellular infiltration, the main components of which were lymphocytes and plasma cells. Organization was occurring and capillary loops and fibroblasts were conspicuous. In the adipose layer of the epicardium there were granular eosinophilic areas, probably representing changes in the ground substance. The myocardium showed scattered areas where muscle fibres had disappeared. There were no distinctive connective-tissue changes. The endocardium and valves showed no abnormalities.

*Respiratory System.*—There was a generalized oedema, but neither vascular nor connective-tissue changes were observed.

*Digestive System.*—The small intestine showed several small vessels with eccentric intimal thickening; similar changes were seen in the mesenteric vessels.

*Haemopoietic System.*—The spleen showed the typical concentric periarterial fibrosis found in systemic lupus erythematosus (Fig. 1).

*Genito-urinary System.*—In the kidneys, the glomeruli showed both "wire loop" lesions and patchy necrotic areas, often associated with crescents. The interstitial tissue was diffusely infiltrated with lymphocytes and plasma cells. Fibrinoid necrosis was present in afferent arterioles and interlobular arteries. Cellular reaction in relation to these was minimal.

*Central Nervous System.*—No vascular changes were found in the brain, although numerous sections were examined. Examination of peripheral nerves in the leg showed that some fibres had disappeared but most of them remained, showing no abnormality with a myelin stain. The endoneurium was increased in amount, varying from bundle to bundle. Abnormalities of the blood vessels in the epineurium were noticed. There were no active lesions such as were seen in the kidney, but numerous vessels showed evidence of previous damage. The arteriole in Fig. 2 shows evidence of old obliteration of the lumen with recanalization, and in relation to it is a group of chronic inflammatory cells. The small artery seen in Fig. 3 shows an eccentric fibrotic thickening in the intimal zone with thinning of the muscular wall over it. The internal elastic lamina is intact and new elastic is present in the fibrous tissue. This vessel has been followed in serial sections, but there was no evidence of previous complete rupture of the wall with loss of elastica and fibrous replacement of the media such as one normally sees in healed periarteritis nodosa. In discussing peripheral neuritis in polyarteritis nodosa, Kernohan and Woltman (1938) described healed vessels in which regeneration of the internal elastica had taken place, and the vessel in Fig. 3 may represent either an organized thrombus or a healed necrotizing lesion with regrowth of the internal elastica lamina. The distribution of the vascular lesions was patchy. Sections taken at certain levels showed completely normal vessels, while those taken at others showed lesions such as have been described.

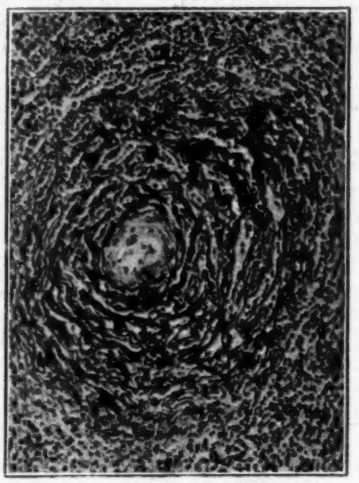

FIG. 1.—Spleen. Concentric layers of collagen surrounding small artery. Haematoxylin and van Gieson. (×110.)

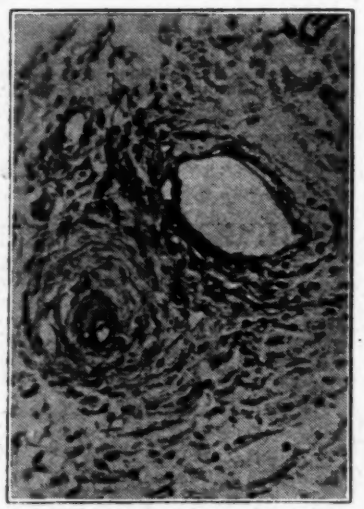

FIG. 2.—Peripheral nerve. Arteriole has been obliterated and recanalized. Chronic inflammatory cells in relation to it. Haematoxylin and eosin. (×180.)

### Discussion

The combination of a prolonged pyrexial illness, renal dysfunction, serous effusions, and central nervous involvement of sudden onset suggested the diagnosis of either systemic lupus erythematosus or polyarteritis nodosa. The skin lesions and the progressive neutropenia were in favour

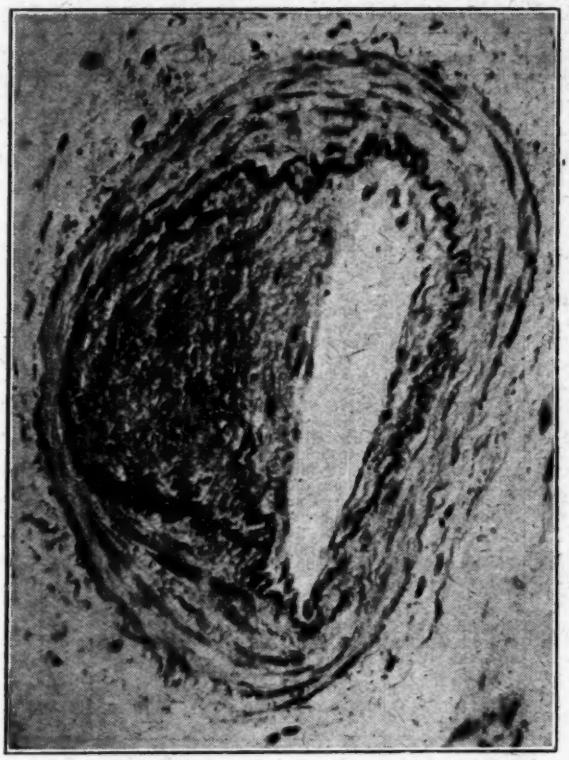

FIG. 3.—Peripheral nerve. Artery with gross intimal thickening and thinning of muscularis over it. New elastic-tissue formation. Hart–Sheridan method for elastic. (×420.)

of the former diagnosis, as was the female sex of the patient. On the other hand, the peripheral neuritis which gave rise to the presenting symptoms suggested polyarteritis nodosa, particularly when no previous account of this lesion in systemic lupus erythematosus could be found.

That this was a case of systemic lupus erythematosus could not be doubted after the post-mortem examination, although there is no satisfactory explanation for the repeatedly positive blood cultures.

The diagnosis of peripheral neuritis is established by the combination of a lower motor-neurone lesion, paraesthesiae, and sensory loss in all four limbs, together with bilateral calf tenderness.

From the histological evidence already given, it is postulated that the neuritis was of a similar nature to that described in polyarteritis nodosa by Wohlwill (1923), due to ischaemic changes in the nerves produced by an obliterative arteritis of the arteria nervorum. In view of the now generally accepted belief that systemic lupus erythematosus is a widespread disease of collagen it is not surprising to find these vessels involved. Indeed, rather is it to be wondered why polyneuritis is not a more frequent manifestation of this disease process.

## Summary

A case of systemic lupus erythematosus in a woman aged 34 is described in which the presenting picture was one of peripheral neuritis.

It is considered that the peripheral neuritis was due to ischaemia consequent on vascular lesions in the vessels supplying the nerves.

We wish to record our indebtedness to Dr. J. W. Litchfield and Dr. P. H. Sandifer, the physicians in attendance on the patient, for their permission to report this case and for the interest they have shown in this paper; to Dr. J. F. Taylor, who carried out the necropsy; and to Mr. A. Beasley for the preparation of countless serial sections. We are most grateful to Professor W. D. Newcomb and Professor G. W. Pickering for their help and advice.

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# A FOLLOW-UP STUDY OF 238 LEUCOTOMIZED PATIENTS*

BY

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This follow-up study was undertaken to assess the results of standard prefrontal leucotomy in 238 patients who underwent the operation at Crichton Royal between 1943 and 1948. Although it may be accepted that the value of the operation is established for chronic obsessional states and chronic affective psychoses, unexpected failures and unexpected successes are often met with. It was hoped that from a study of our own patients there would emerge criteria that would help us to select those individuals likely to respond to the operation. A special interest was taken in schizophrenics because they made up more than three-quarters of the series and because they form the great therapeutic problem. Partridge (1950) has published a valuable book on his personal follow-up of 300 patients over a period of one and a half to three years after operation; he was more interested in the clinical appearances than in the tabulation of results. We, on the other hand, to avoid being influenced by clinical impressionism have made a detailed examination of our material and results.

## Material

TABLE I.—*Composition of the Clinical Material*

| Diagnosis | No. of Cases | Percentage of Cases |
|---|---|---|
| Schizophrenia .. .. .. | 194 | 81·1 |
| Affective psychoses .. .. .. | 21 | 8·9 |
| Obsessional neurosis .. .. .. | 14 | 6·3 |
| Mainly organic psychoses .. .. | 9 | 3·7 |
| Total .. | 238 | 100 |

Important features of our series were that all the patients were diagnosed and selected for operation according to the uniform standards of our hospital, while with the exception of the first 16 patients all operations were carried out by the same surgeon. The diagnosis in the 238 cases is shown in Table I. Most of the patients received the standard coronal cut of Freeman and Watts, but in 52 chronic cases

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the incision was varied so as to isolate the orbital lobes and to spare the upper quadrants; this modification was abandoned because the results were less good (Egan, 1949).

The mortality rate from the operation in our series was 3%. There were none of the "delayed post-operative deaths" described by Meyer and McLardy (1948). The incidence of post-operative epilepsy, including single fits, was 8%, most of these being male catatonic schizophrenics. There were no other serious organic complications, and persistent enuresis was not encountered.

*Selection of Patients.*—Our criteria of selection have always been conservative, so that only 13 of our patients had been ill for less than two years. The operation is not considered until full courses, often repeated courses, of the appropriate physical treatments have failed to induce a sustained remission. Each case was discussed pre-operatively at a staff conference, and if we thought there was any reasonable hope of remission without operation then leucotomy was avoided. Our criteria for selection included a good pre-psychotic personality, good retention of personality, and the presence of emotional tension.

## Method of Follow-up

The method of follow-up we adopted for discharged patients was by personal letter to informants whom we knew to be reliable. These informants were either the relatives, the family physician, a psychiatric social worker, or, in the case of hospital patients, the appropriate medical superintendent. In some cases we also wrote to the patient himself. Our letter put the following specific questions: (1) Is the patient fully and satisfactorily occupied? (2) Is he happy and his old self again? (3) Is he easy to get on with and does he take part in social life? (4) Are there any special difficulties? (5) Do you find him a different person from what he was before his illness began?

The first inquiry elicited satisfactory information in a great majority of cases, and at the end of our investigation only one of the 238 patients was untraced. A postal follow-up has disadvantages; so has a personal interview, because the patient can give a deceptive impression of normality, and this has to be corrected by more objective opinions from relatives and doctors. We are satisfied that our study gives an accurate picture of the leucotomized patient's place in society, and, after all, criteria of success or failure after leucotomy are mainly social criteria. It was found that the patients' own letters reflected an uncritical self-satisfaction and lack of judgment, while they were prone to describe ease of social relations when in fact their social adaptation was impaired.

The average time between operation and our inquiry was four and a half years; the shortest was one year four months and the longest seven years two months. There is no doubt that follow-up studies made less than one year after operation give a deceptive picture of the results of leucotomy.

## Results

The patients' condition during the first four months of 1950 is indicated in Table II. From this we see that, broadly speaking, of these prognostically hopeless cases almost one-third are living a normal or almost normal family life, and more than one-fourth are usefully and gainfully employed. Of those who remained in hospital almost half are much improved; they are happier and are easier to nurse. Our findings over a lengthy period confirm, therefore, the findings of those workers who have followed their cases for only a short time.

Apart from assessment of the therapeutic results of the operation, an attempt was made to discover which factors influenced these results.

## Factors Influencing Results

*Sex.*—Although, like other workers, we have found a greater number of good results in females, our figures are not significant. However in schizophrenia this same trend has been found by others in spontaneous remissions (Guttmann, Mayer-Gross, and Slater, 1939) and also in those who received insulin coma treatment (Mayer-Gross, 1951, unpublished observations). It may in part be correlated with the greater ease with which a female can be again accepted into family life.

TABLE II.—*Graded Condition of Patients at Time of Investigation*

| Grade | Condition | Definition | No. | % |
|---|---|---|---|---|
| I | Recovery | Social life as before illness. Working at old job or at one at the same occupational level as before illness. Minimum of any form of symptoms | 41 | 17·2 |
| II | Social recovery | Relatively normal social life. Lower standard of work; may or may not be gainfully employed. Some symptoms of psychosis or marked post-leucotomy symptoms present | 31 | 13·0 |
| III | Social defect | Deterioration in social behaviour, sometimes even antisocial. May or may not be gainfully employed. Symptoms as with social recovery | 15 | 6·3 |
| IV | Home invalid | Require care and observation in their daily life, and in any simple occupation which they may do at home. Not otherwise employed. Marked symptoms still present | 15 | 6·3 |
| Va | Hospital invalid | Generally improved—i.e., able to work in hospital and take part in entertainments. Have some form of parole | 21 | 8·8 |
| Vb | Hospital invalid | Easier nursing problems; no longer so aggressive and impulsive. Some may have a simple occupation in the ward | 20 | 8·4 |
| Vc | Hospital invalid | No change since operation | 22 | 9·3 |
| Vd | Hospital invalid | Deteriorated | 28 | 11·8 |
| VR | Readmitted to hospital, and in at April, 1950 | | 35 | 14·7 |
| VI | Dead | As a result of operation | 7 | 3·0 |
| | | From other causes | 2 | 0·8 |
| VII | Untraced | | 1 | 0·4 |
| | | Totals | 238 | 100 |

*Age at Operation.*—Good results occurred equally in our three age groups: (*a*) those under 30 years; (*b*) those of 30–39; and (*c*) those of 40 and over. However, the oldest age group had the smallest number of relapses.

*Pre-morbid Personality.*—By examining the case records and social histories we classified our patients into those who had been well adapted to life and society before the psychosis appeared and those whose pre-morbid adaptation had been poor and unstable. Of those who had a well-adapted pre-morbid personality 68% made grade I or grade II recoveries. Of those who were poorly adapted before their illness only 35% made grade I or II recoveries.

*Time Spent in Hospital.*—By this we mean the total of time in hospital since the illness began. Our total of grade I and II recoveries was 50% of those who had been in hospital under one year and only 11.5% of those who had been in hospital more than five years.

*Duration of Illness.*—By this we mean the total time since symptoms first became apparent. Here our findings were unexpected and we think important. In the group of patients who had been ill for 10 years and over there were almost as many grade I and II recoveries as there were in the group who had been ill less than five years (see Table III).

TABLE III.—*Relationship Between Duration of Illness and Condition at Time of Investigation*

| Duration of Illness (years) | Grade (see Table II) | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | I | II | III | IV | Va | Vb | Vc & Vd | VR | VI | VII | Totals |
| Under 5 .. | 14 | 11 | 2 | 6 | 6 | 4 | 12 | 18 | 3 | 1 | 77 |
| 5–9 .. .. | 14 | 9 | 8 | 3 | 6 | 7 | 15 | 11 | 3 | 0 | 76 |
| 10 and over .. | 13 | 11 | 5 | 6 | 9 | 9 | 23 | 6 | 3 | 0 | 85 |
| Totals .. | 41 | 31 | 15 | 15 | 21 | 20 | 50 | 35 | 9 | 1 | 238 |

The conclusion is that, by itself, long duration of illness should not weigh against performance of the operation. Freeman and Watts (1950) make this same point. The apparent discrepancy between the effects of length of illness and length of stay in hospital is probably to be explained by the view that the patient who is able to remain outside hospital, even though ill, does so because of his good retention of personality and his social adaptability.

*Retention of Personality.*—By this we understand that, despite his psychosis, the patient retains an interest in his appearance, in personal and topical affairs, and in social intercourse. Of those patients with good pre-operative retention of personality 46% made grade I or II recoveries. Of those who seemed deteriorated before operation 21%, less than half, made grade I or II recoveries. Even this 21% is quite a respectable figure, but then it is notoriously difficult, especially in schizophrenia, to determine the presence or absence of deterioration. We found that probably the best test is the response to electric convulsion treatment (E.C.T.), and we would now hesitate to stigmatize a patient as too deteriorated for operation until we had seen the response he made to a course of electroplexy.

*Response to Electric Convulsion Therapy.*—Of those who had made a good, albeit transient, response to E.C.T. given before leucotomy 22% made grade I recoveries, while of those who had shown no response to E.C.T. only 3.5% made grade I recoveries. Table IV illustrates these results.

TABLE IV.—*Relationship Between Response to E.C.T. and Condition at Time of Investigation*

| Clinical Response to E.C.T. | Grade (see Table II) | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | I | II | III | IV | Va | Vb | Vc & Vd | VR | VI | VII | Totals |
| No improvement | 2 | 11 | 6 | 2 | 5 | 5 | 16 | 8 | 2 | 0 | 57 |
| Temporary improvement | 33 | 19 | 8 | 13 | 13 | 13 | 23 | 22 | 6 | 1 | 151 |
| No E.C.T. given | 6 | 1 | 1 | 0 | 3 | 2 | 11 | 5 | 1 | 0 | 30 |
| Totals .. | 41 | 31 | 15 | 15 | 21 | 20 | 50 | 35 | 9 | 1 | 238 |

*Response to Insulin Coma Therapy.*—On the other hand, we found no significant differences after leucotomy between those schizophrenics who had made some response and those who had made no response to insulin coma therapy.

*Emotional Tension.*—It is generally agreed that the best results of leucotomy occur in those patients who still show a strong affective reaction to their psychosis. As the presence of this affective reaction or emotional tension was one of our main criteria of selection, there were only 16 patients in the series who showed no obvious tension: the figures are therefore too small for comparison. It is interesting, however, that even of these 16 patients five made grade I or II recoveries.

*Diagnostic Categories.*—The series included 35 cases of affective psychosis and obsessional neurosis and 194 cases of schizophrenia. In the former group 60% made grade I or II recoveries, while only 27% of the schizophrenics reached these grades. Surprisingly, all our five cases of mania made grade I or II recoveries.

*Relapses.*—It was found that of 115 patients who had improved enough to be discharged from hospital 22 relapsed after an average period of two and a quarter years free from symptoms. When this group of relapsed cases was compared with those who had maintained their recoveries the only significant factor found was that the pre-operative personality was more often intact in the recovered cases than in the relapses (Gillies and Mayer-Gross, 1950).

### Summary and Conclusions

A follow-up study was made of 238 chronic psychiatric patients who had undergone the standard leucotomy operation some one to seven years previously. The best results occurred in those whose pre-operative personality was more or less intact, who had well-adapted pre-psychotic personalities, and who had been only a short time in hospital. If these factors were present, long duration of illness did not worsen the prognosis.

In schizophrenia the best test for pre-operative deterioration is afforded by the response to electric convulsion treatment.

Of the psychiatric patients who have hitherto been regarded as hopeless hospital invalids the standard leucotomy can return one-third to a normal or almost normal social life and can fit one-quarter for gainful employment.

Broadly speaking, the prognostic criteria for leucotomy are still those well-tried criteria that hold for the untreated psychosis.

We are indebted to Dr. P. K. McCowan for his advice and for permission to publish the material; our thanks are also due to Mr. R. L. Beveridge, who operated on most of our patients.

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# FAMILIAL ERYTHEMA NODOSUM

BY

**JOHN FRY, M.B., F.R.C.S.**

*(From a General Practice)*

Erythema nodosum is a clinical entity with a characteristic skin eruption, usually accompanied by a general constitutional upset. Diagnosis of the eruption is easy, but there is still some uncertainty with regard to the aetiological aspects of this condition. The general consensus of opinion is that it is a non-specific reaction in a sensitive subject in response to a number of various allergens. These are best discussed under separate headings.

*Tuberculosis.*—It is probable that erythema nodosum is most often associated with the primary tuberculous complex, particularly in children and adolescents. In these cases the eruption occurs when the patient first develops hypersensitivity to tuberculin. The time-interval for this to happen has been estimated at three to eight weeks from the time of infection. The frequency with which erythema nodosum is associated with primary tuberculosis apparently varies individually and also nationally. Thus Wallgren (1938) estimated that 30% of primary infections in Scandinavia were associated with the eruption; Dunlevy (1949), in Eire, found only a 2.8% incidence. In Scandinavia the average age of primary infection is much higher than that in Eire. Wallgren, in a series of 800 cases, reported that 95% had a positive tuberculin skin reaction. This is significant, because these Scandinavian children could normally have been expected to have a negative reaction. He also describes a number of cases in which the change of the skin response from negative to positive was definitely associated with the eruption.

Punch (1941) was able to show that in student nurses the change from a tuberculin-negative to a tuberculin-positive skin reaction was often accompanied by an eruption of erythema nodosum. Bruce Perry (1944), in a series of 112 cases, found a positive Mantoux reaction in 54%. In those under 15 the incidence was 75% and in those over 15 it

was only 25%. Doxiadis (1949) found 88% tuberculin-positives out of 100 children under 15 who developed erythema nodosum. There are relatively few reports on series of adult cases. Löfgren (1946) considers that a non-tuberculous cause is more frequent in adults.

*Acute Rheumatism.*—The usual view held in this country, until recently, was that erythema nodosum was a frequent accompaniment of acute rheumatism. All the latest papers discussing the aetiology of erythema nodosum stress the rarity of this association (Wallgren, 1938; Bruce Perry, 1944; Löfgren, 1946; Doxiadis, 1949). The points against the rheumatic theory are the absence of any signs of carditis in erythema nodosum, the different sex and age incidence, and the lack of response to salicylates. Although rheumatic fever as a common cause is discredited, it is certain that erythema nodosum may occasionally be a manifestation of acute rheumatism.

*Streptococcal Infection.*—It was Collis (1932) who first drew attention to the fact that tuberculin-negative cases of erythema nodosum had a markedly higher sensitivity to streptococcal nucleoprotein at the time of the eruption. It seems probable that erythema nodosum may be an expression of streptococcal allergy in certain cases. It is from this cause that cases of recurrent attacks occur. Rotnes (1936) reported a case in which there had been 14 attacks, usually after a pharyngitis or tonsillitis.

*Sarcoidosis.*—Erythema nodosum is occasionally met with as a sign of sarcoidosis (Kerley, 1942, 1943; Löfgren, 1946; Crawley, 1950).

*Sulphonamide Hypersensitivity.*—In the early days of sulphonamide therapy skin rashes of diverse types were common. At the present time, with compounds of lower toxicity, these rashes are much less frequent. Occasionally an eruption indistinguishable from erythema nodosum is found in these circumstances, and it disappears rapidly when the drug is stopped. It is difficult to know whether the eruption is the result of the primary condition or whether it is a complication of the treatment.

*Coccidioidomycosis.*—For many years a chronic granulomatous condition, due to infection by *Coccidioides immitis*, has been prevalent in the San Joaquin Valley in California. This condition has a number of features akin to tuberculosis. There is a mild primary stage of infection which is sometimes followed by a general blood-stream spread and a possible late occurrence of chronic granulomata in the lungs and other organs. About 5% of these cases have a typical eruption of erythema nodosum associated with the primary stage of infection.

*Miscellaneous.*—There are a number of other conditions with which erythema nodosum has been associated—for example, meningococcal, gonococcal, and staphylococcal septicaemias; lymphogranuloma inguinale; syphilis; lymphadenoma; and intoxication with bromides and iodides.

*Question of Specificity.*—The recognition of small epidemics of erythema nodosum gave rise to the theory that it is a specific disease (J. O. Symes, 1921; Lendon, 1925). The causal organism has, however, never been convincingly isolated. Further studies of these epidemics suggested that in the majority there was evidence of a recent simultaneous contact with the tubercle bacillus.

Three cases of erythema nodosum which occurred simultaneously in one family are described.

## Case Reports

The family, in October, 1950, comprised the parents and three young children: Mary aged 7½, Janet 5, and Roger 3½. They lived in a house of three bedrooms on the outskirts of South-east London. The parents slept in one bedroom, the two girls shared a double bed in the second, and Roger had a small room to himself. The father, a builder's decorator aged 30, had been unwell for some weeks with a cough, and on October 21 bilateral open pulmonary tuberculosis was diagnosed. He was at once ordered to bed. The mother then slept with her two daughters in their double bed, and the father had a separate room. He remained there until November 29, when he was admitted to a sanatorium. When tuberculosis was diagnosed the rest of the family were investigated at the local chest clinic. Chest radiographs showed no signs of any disease, and tuberculin tests on the children were all negative.

On December 1 the mother was seen on account of a sore throat, of three days' duration, and a painful eruption on her legs, which she had noticed for 12 hours. She was a healthy young woman of 28. She had a clean medical record, and was at this time four months pregnant. There were no cough, joint pains, or any digestive upset. The whole family had had a meal of prawns on the previous day. Examination at this stage revealed a normal temperature—98.2° F. (36.8° C.)—and pulse rate 81. The fauces were hyperaemic, but there was no exudate. On the extensor aspects of both legs there were about a dozen typical areas of erythema nodosum. Over the next four days her general condition became a little worse, with a fever up to 100° F. (37.8° C.), and increasing pain in the legs. On the fifth day she began to improve, and the eruption had disappeared by the tenth day. She received no specific treatment.

On December 1 Mary, aged 7½, began to feel unwell with vague dull mid-abdominal pain and fever—99.5° F. (37.5° C.)—and a pulse rate of 92. She showed no abnormal physical signs at this stage. There was no sore throat, no vomiting or diarrhoea, or any joint pains. On December 4 the characteristic eruption of erythema nodosum appeared on the anterior aspects of both legs. By this time she felt better and there was no abdominal pain or other constitutional upset. The eruption persisted for eight days. She, too, had no specific therapy.

Janet, aged 5, was quite well in spite of the appearance of typical areas of erythema nodosum on her legs on December 6. There was no fever or tachycardia. Her eruption had disappeared within seven days.

Roger, aged 3½, had during all this time remained in his usual good health and continued to do so.

All four were further investigated. Chest radiographs of the mother, Mary, and Roger on December 21 were considered to be within normal limits. Janet's radiograph showed a slight degree of left hilar enlargement. Further radiographs on June 4, 1951, were all regarded as within normal limits. A tuberculin patch test was performed on all four on December 15, and Janet showed a positive response. A Mantoux, 1/1,000, however, resulted in a positive reaction in the other three. The family has remained well since December, 1950, until the time of writing (July, 1951). The mother was delivered of a normal healthy boy in April, 1951; he is Mantoux-negative (1/1,000), and is about to have B.C.G. vaccinations. Janet and Roger had measles in March, but have otherwise remained well. The father is still in a sanatorium, but is due for discharge soon.

## Discussion

It seems certain that in the two girls the development of a positive tuberculin skin reaction accompanying a primary tuberculous infection was associated with an eruption of erythema nodosum. This is also likely to be the case in the mother, but it is inconclusive, as a previous skin test was not done.

A number of previous reports describe similar series of cases. The most remarkable of these was an "epidemic" which occurred in the Welsh town of Mynyddislwyn in the summer of 1920. It is reported by J. O. Symes (1921), quoting R. E. Roberts, who was then the local M.O.H. Over a period of five weeks 50 cases of erythema nodosum were seen in an area enclosing three streets. There had been no cases in this town for the previous 15 years. There is no mention of any search for a tuberculous cause. W. L. Symes (1907) reported 12 cases occurring over a period of four weeks in a girls' residential school of 100 pupils.

Robinson (1926) described three cases. Lendon (1925), in a survey, quoted 10 instances of outbreaks of erythema nodosum; in two of these there was a subsequent tuberculous infection. Demole (1933) reported three cases in a dressmaking school where a tuberculous source was found. He also states that in the nineteenth century there were widespread epidemics of erythema nodosum in Bosnia, Jutland, and Turkey.

Wallgren investigated an outbreak which occurred in a class of 34 girls: 18 suffered simultaneously from a febrile condition. On follow-up 12 of these subsequently developed erythema nodosum within 6 to 10 weeks. Three months previously there had been an open case of pulmonary tuberculosis in the class. Further small series with a tuberculous aetiology are reported by Lannois (1892), Ledoux (1932), Parisot and Saleur (1933), Bertoye and Bernheim (1935), and Mouriquand and Weill (1937). Cibils Aguirre (1933) has collected a number of series of "familial erythema nodosum" cases with a probable tuberculous cause.

It seems likely that most of these multiple cases of erythema nodosum have been associated with a primary tuberculous infection. It is true that a definite tuberculous contact has not been found in some of these, but perhaps the contact was of a transient and elusive nature.

**Summary**

The possible aetiology of erythema nodosum is discussed. It is a non-specific reaction to a variety of allergens. The most important of these is the association of erythema nodosum with a primary tuberculous infection.

A report is given of three cases occurring simultaneously in one family. In these there was a definite tuberculous cause.

Previous reports of similar cases are reviewed.

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According to the Hungarian News and Information Service the first Congress of the Hungarian Microbiological Society was held in Budapest during January, and was attended by 250 Hungarian scientists, and 26 scientists from eight other countries, including the Soviet Union, China, and Korea. Eighteen lectures were delivered to plenary sessions of the Congress, and nearly 150 to meetings of its eight sections. The society was formed during 1951, and the congress was the first public presentation of the work of Hungarian microbiologists. Dr. Károly Rauss is engaged on research into a preventive vaccine for dysentery. Professor Rezsö Manninger is working on vaccines against fowl pest, swine fever, and other animal diseases. Typhoid fever carriers, antibiotics, and the manufacture of penicillin are all being investigated. The Parasite Section of the National Institute of Public Health is working on the prophylaxis of malaria. Messrs. Kardoş, Kiss-Bagoly, and Deutsch discussed the question of the formation of bacteria from non-cellular protein.

# A NEGATIVE BENZODIOXANE TEST IN PRESENCE OF A PHAEOCHROMOCYTOMA

BY

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Four cases of phaeochromocytoma have been reported which gave a negative response to the benzodioxane (piperoxane) test before operation. Prunty *et al.* (1950) described the case of a man, under the care of Professor Pickering, in whom a large phaeochromocytoma was removed at operation, despite a pre-operative negative benzodioxane test. Smithwick's case, described by Goldenberg and Aranow (1950), was that of a middle-aged woman with persistent hypertension and a negative benzodioxane reaction who was found to have a large phaeochromocytoma at operation. Calkins and Howard (quoted by Goldenberg and Aranow) described a similar case. Beyer *et al.* (1951) reported a negative benzodioxane test in a woman aged 40 in whom a phaeochromocytoma of the left adrenal was removed at operation.

The following case report concerns a woman with sustained hypertension and a negative benzodioxane reaction, who at necropsy was found to have a phaeochromocytoma.

## Case Report

A woman aged 25 was admitted in September, 1950, for gynaecological investigation following a prolonged history of menstrual disturbances, anorexia, and loss of weight, with associated attacks of blurred vision and frequent night sweats. Routine examination revealed a sustained hypertension ranging from 240/130 to 220/120 and a persistent tachycardia (88–120), although she was a psychologically stable patient. She had a large hypertensive heart but no congestive lung changes. Retinoscopy revealed gross papilloedema with haemorrhages, exudates, and attenuated arteries.

*Investigations.*—Sodium amytal test (see Fig. 1). The benzodioxane test, carried out as described by Goldenberg, with a calculated dose of 17 mg., gave a negative result (Fig. 2). An electrocardiogram showed gross left ventricular stress. Renal function tests showed a normal blood urea; albumin, red cells, and granular casts in all urine specimens; and imperfect function. An intravenous pyelogram was normal.

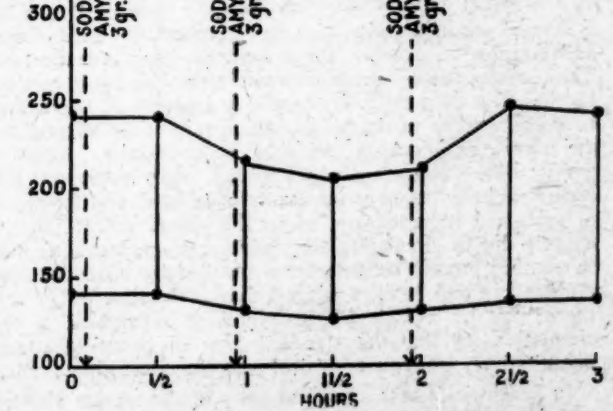


FIG. 1.—Graph showing results of the sodium amytal test.

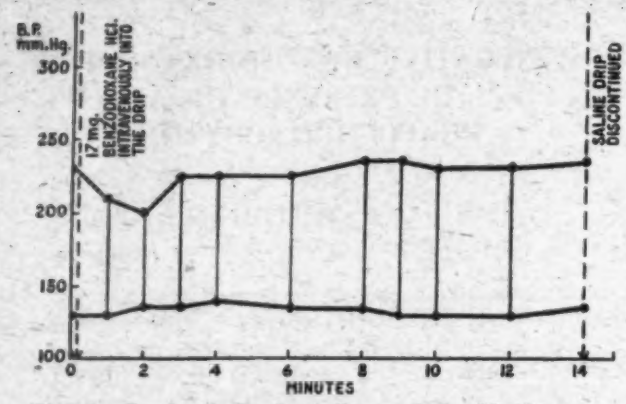


FIG. 2.—Graph of the benzodioxane hydrochloride test.

The patient was given a course of intramuscular hexamethonium bromide without result, and subsequently a bilateral lumbo-dorsal sympathectomy was carried out by Mr. J. R. Ratcliffe. No tumour was palpated at operation. A week after the second stage of the operation she developed total blindness, with gross retinal oedema, and her blood pressure remained persistently high. The blood urea never rose above 40 mg. per 100 ml. Death occurred five weeks after the operation. Necropsy, performed by Dr. Ian Mackenzie, showed a large hypertensive heart, with renal changes consistent with chronic nephritis. A large phaeochromocytoma of the right adrenal was found, hidden deep under the right lobe of the liver.

## Discussion

The use of the benzodioxane test was first described by Goldenberg *et al.* (1947), who stated that if the hypertension is due to an adrenaline- and *nor*adrenaline-producing tumour, a drop of 20–50 mm. in both systolic and diastolic pressures occurs within four minutes, with a return to normal in 15 minutes. The result in the above case was therefore taken to be negative.

Certain features of the case suggested the necessity for a benzodioxane test. There was no history of pregnancy toxaemia or chronic nephritis, though the latter is an admittedly difficult differential diagnosis in the presence of signs of renal disease. Malignant hypertension is uncommon at this age in the absence of these two causes. The attacks of nocturnal sweating, persistent hypertension, tachycardia, and gross retinal changes aroused suspicion of a phaeochromocytoma.

It is suggested by Beyer *et al.* that *nor*adrenaline is the immediate precursor of adrenaline in the body, and that in phaeochromocytoma there is an impairment of the transmethylation reaction, so that, as other observers have found also, the content of *nor*adrenaline in the tumours may be 50 to 90% of the total pressor substances. The greater the preponderance of *nor*adrenaline the greater the probability of a negative benzodioxane reaction.

Several theories have been put forward by Goldenberg and Aranow to account for a negative benzodioxane test. They assume that a "secondary mechanism" is set in motion by the effect of persistently excessive amounts of adrenaline or *nor*adrenaline in the blood, which fails to be affected by the adrenergic blocking action of benzodioxane. Alternatively, this "secondary mechanism" may represent the "vicious circle" of experimental hypertension, and the phase of sustained hypertension, as in the above patient, is not entirely due to circulating adrenaline and *nor*adrenaline, but to another pressor substance not blocked by benzodioxane. Goldenberg and Aranow suggest that at the time of the test there may be a lack of these pressor substances in the circulation, or that the standard dose of benzodioxane is quantitatively inadequate to deal with the circulating amines.

In Smithwick's case, chemical assay of the tumour showed no abnormal pressor substance which would be resistant to the effects of benzodioxane. These occasional negative responses to benzodioxane in the presence of a phaeochromocytoma should not diminish the use of the test at the bedside.

If the test is negative or doubtful it should be repeated, particularly when other investigations suggest a tumour. It is unfortunate that the most reliable test—that by the injection of histamine intravenously as suggested by Roth and Kvale (1945)—is too hazardous for routine use. Repeated straight radiographs of the abdomen are a valuable procedure in certain cases. The insufflation of perirenal air, while a satisfactory procedure in some cases, can be dangerous in other than expert hands.

Whether such a procedure in the above case would have shown up the tumour is doubtful because of its position deep under the right lobe of the liver. Urinary assays of adrenal medullary hormone derivatives, as introduced by Engle and von Euler (1950; see also von Euler, 1951), are not yet readily available for use in diagnosis, though, if confirmed, their methods will provide considerable diagnostic certainty.

When a lumbo-dorsal sympathectomy is being performed the kidneys and adrenals should be palpated for a tumour, though in my case no tumour was detected.

A diagnostic laparotomy may be necessary occasionally, but hyperadrenalism may develop when the tumours are palpated, with acute cardiac failure supervening on an already overstrained cardiovascular system for hyperadrenalism. Thus the tumour should be removed at the initial operation (Hubble, 1951).

## Summary and Conclusion

A case of phaeochromocytoma is described in a young woman of 25, in which a negative result to the benzodioxane test was obtained. Four similar cases from the literature are briefly reviewed, and the possible reasons for failure of the test are discussed. If the test is doubtful or negative when a phaeochromocytoma is strongly suspected it should be repeated, and other diagnostic measures undertaken.

I am indebted to Dr. Douglas Hubble, physician, Derbyshire Royal Infirmary, for much helpful and constructive criticism in preparing this report. I would also like to thank Dr. Ian Mackenzie for his most valuable necropsy report.

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Under Service conditions there is a demand by military patrols for a convenient method of sterilizing small quantities of water in individual water-bottles. During the 1939–45 war when necessary a white tablet of "halazone" (*para*-sulphon-dichloramido-benzoic acid) was placed in the water-bottle filled with water and shaken vigorously to form chloramine. After 30 minutes one blue tablet (coloured sodium thiosulphate) was added and the bottle again shaken to remove the taste of the halazone tablet. It was found that these tablets deteriorated in humid tropical climates, becoming adherent to one another, and producing only small amounts of chloramine. New tablets have been produced using chloramine B (sodium-chlorobenzene-sulphonic acid amide), which has stored well for over two years in various theatres overseas, though unfortunately the tablet becomes hard and takes a long time to dissolve. With the new tablet detasting will be unnecessary, and the contact time for sterilization will be reduced to 15 minutes.

# Medical Memorandum

## Parkinson's Syndrome following Severe Herpes Ophthalmicus

A number of papers have recently appeared in various journals on the complications of herpes zoster. These are lesions of motor neurones, myelitis and meningitis, and encephalitis. Alajouanine and Griffith (1931) record a case of a girl of 17 who developed typical signs of disseminated sclerosis two years after a "herpes myelitis." The following case of Parkinson's syndrome occurred after severe herpes ophthalmicus.

### CASE REPORT

A flight lieutenant aged 48 had a healthy Service record of nearly 30 years, apart from gastric symptoms spread over a number of years. He first noticed slowness of movement and marked general tremor in January, 1949. There was no history of varicella, but he thought he had had measles and mumps. In 1941 he was bombed, and his hands and face were burnt; a duodenal ulcer developed about four weeks later. He had no residual effects. In May, 1947, he contracted severe left herpes ophthalmicus with corneal ulceration, from which he took a long time to recover. He had felt ill and thought he had been febrile. The left side of his face was "dead" for some months, and an operation had been performed on the left lower lid. There was no history of any illness like encephalitis lethargica. He was not vaccinated or immunized either before or after his attack of herpes.

In November, 1949, he gave the typical picture of Parkinson's syndrome—mask-like facies, slow movement, festinant gait, tremor of both hands, stiffness of face and neck muscles, and slow speech. Rigidity of arms and legs with increased tendon-jerks was present. Examination of the central nervous system, chest, and abdomen showed nothing abnormal.

*Investigations.*—A radiograph of the skull and lumbar puncture were normal, and the Wassermann reaction was negative. Psychometric tests showed signs of cortical atrophy due to arteriosclerosis. His blood pressure was 138/86. His urine was normal.

The patient had had belladonna and hyoscine hydrobromide and then was given "artane" (trihexyphenidyl), 15 mg. daily at first, finally increased to 17 mg. daily. Relief of depression was the first symptom alleviated, then diminution of sialorrhoea; tremor was not so pronounced, but spasticity was not greatly relieved. He was to have had other drugs, when he was found dead in bed on September 14, 1950.

At necropsy no definite lesion was found that could account for death. His coronary and cerebral vessels showed no evidence of arteriosclerosis, but were in a state compatible with that of any other man of his age. Analysis of organs was negative. Death was recorded as cardiac failure due to Parkinson's syndrome. Sections of the brain stem and basal ganglia were not helpful.

### COMMENT

There appeared to be three possibilities: (1) That herpes ophthalmicus was in no way connected with the subsequent lesion. (2) That herpes ophthalmicus was a precipitating factor. (3) That the subsequent lesion or lesions were due absolutely to the effects of the virus. Obviously, the second and third possibilities must be examined before accepting or rejecting the first. There was no history of any condition following the herpes ophthalmicus which would suggest a zoster encephalitis or encephalitis lethargica. The virus responsible for herpes belongs to the group which produce degenerative changes in the nerve cells, and has also been termed the sensory analogue of anterior poliomyelitis. In the latter, encephalitis, myelitis, and brain-stem lesions may occur; in the former, encephalitis and cord lesions have occurred; brain-stem lesions are therefore within the realms of possibility.

Instead of regarding the virus as acting locally Head and Campbell (1900) put forward the view that the initial febrile phase indicated a general systemic infection; the evidence now accumulating suggests that there is a stage in which the virus is disseminated throughout the nervous system. Magnusson and Wohlfart (1942), on anatomical examination in a case of herpes zoster ophthalmicus, found slight general meningitis and herpetic changes in the Gasserian ganglion on *both* sides, though they were most pronounced on the side of the vesicles. Lhermitte and his colleague have shown that even in uncomplicated cases herpes zoster produces lesions in the posterior root ganglia and also causes inflammation of the anterior horns of the segments of the cord relative to the affected ganglia. Again, Lhermitte and Vermès (1930) describe lesions in the brain stem and basal ganglia which they found themselves and which were due to the virus of herpes zoster. In these the vessels are tortuous and distended with blood, there are scattered haemorrhages which, though small, are numerous, and areas that appear translucent on section owing to the presence of lacunae, particularly in the region of the pyramidal tracts. Although the patients were advanced in age the authors consider that the lesions are directly due to the herpes virus, since they are similar in type to lesions in the cord, and do *not* resemble lesions due to arteriosclerosis or thrombosis.

Gordon and Tucker (1945) mention a case in which a right hemiplegia with an aphasia developed three weeks after herpes of the ophthalmic division of the trigeminal nerve. The patient, aged 26, had no evidence of arteriosclerosis elsewhere. They suggested a lesion in the brain stem or basal ganglia rather than a localized haemorrhagic or destructive focus affecting the left pyramidal tract in its course between the cortex and mid-brain. Denny-Brown, Adams, and Fitzgerald (1944) support the observation that herpes zoster in the course of generalized invasion may lead to inflammatory thrombosis or haemorrhage.

While the patient remained alive it could be argued that his Parkinson's syndrome was due to arteriosclerosis; this was supported by a psychometric test. The post-mortem examination removed this objection.

In the absence of definite histological findings the evidence presented allows a reasonable assumption that the virus or effects of the virus were responsible: his age; the absence of cerebral arteriosclerosis; the absence of any illness like encephalitis lethargica; and the knowledge that herpes zoster is not necessarily a benign infection, and the relatively high dosage of artane while he was alive.

My thanks are due to Ministry of Pensions Medical Services Division for the results of investigations, and to Dr. R. B. H. Tierney, pathologist.

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# Reviews

## TEXTBOOK OF OBSTETRICS

*Textbook of Obstetrics.* By John F. Cunningham, M.D., M.A.O., F.R.C.P.I., F.R.C.O.G. (Pp. 499; 297 illustrations. £2.) London: William Heinemann. 1951.

Professor John Cunningham, of University College, Dublin, has written this textbook for students and young graduates. The book is well printed, with many clear headings, and profusely illustrated; yet with its 487 pages of text it is small enough to be easily handled. The writing is clear and to the point, yet gives the reader no impression of being deliberately condensed. A remarkable amount of material is contained in the pages. The author mentions Sheehan's work on anterior pituitary necrosis; Weiner's, Race's, and Fisher's contributions to the subject of erythroblastosis foetalis; the use of penicillin and streptomycin; and much recent work. He finds space for brief historical notes on subjects such as the history of the obstetric forceps, but gives no references to literature.

The illustrations by Dr. T. MacDonald and Dr. O. J. Murphy deserve special mention. Most are crude line drawings, yet they undoubtedly convey the exact impression intended, no more and no less—a merit not always found in more ambitious artistry.

The teaching, although orthodox, is free from the trammels of tradition. In discussing the treatment of eclamptic fits the author states that "chloroform, although much used in the past, is now discarded as too toxic." The puerperal patient is to be "treated as a convalescent from the second day. She may be allowed out of bed to use the commode (placed conveniently to the bed) even when there are many sutures in the perineum." Exercises for the puerperal woman are commended, and "should be done gracefully and gently."

When so wide a field is covered with such commendable brevity criticism is inevitably invited; but in this case criticism can only be benign. More emphasis might be laid on the possibility of rupture of the uterus after classical caesarean section and particularly on the fact that rupture in these cases is often "silent," or nearly so, in its early stages. The advice on the management of breech delivery on p. 321 might be questioned:

> "If the breech, whether complete or frank, fails to enter the pelvis, after the second stage of labour has been in progress for a reasonable time with good uterine contractions, disproportion due either to a small pelvis or to a large foetus is the most likely cause . . . . If the disproportion is slight or absent, one or more legs should be brought down and labour allowed to proceed."

To the inexperienced practitioner faced with a case in which labour has advanced into the second stage, and in which the breech has still not engaged in the pelvic brim, this counsel might well lead to danger.

The illustrations have already been favourably mentioned. Fig. 150 might usefully be redrawn—or a new figure included—to show the retroverted gravid uterus in the incarcerated state; the existing illustration does nothing to impress on the student the essential features of elevation of the cervix, elongation of the urethra, and distension of the bladder. Figs. 280 and 288 might both be improved; in the first it is doubtful if the "light" grip of the head would enable the operator to rotate the occiput to the front; in the second the positions of the head and of the forceps in relation to the lower-segment caesarean incision are misleadingly oversimplified.

Professor Cunningham has been at pains to present the methods of treatment in obstetrics "in accordance with the Ethical Standards of the Roman Catholic Church." Thus, in discussing the treatment of hydrocephaly, he advises that the head should be tapped with a spinal or long serum needle. "There is no ethical objection," he states, "to employing this treatment as the operation does not kill the infant." This counsel may not be understood or accepted by all readers, but the ethical considerations will be respected by all. Needless to say, the operation of sterilization receives no mention on any page.

This book is a short and eminently readable exposition of the essentials of the theory and practice of obstetrics. I like it.

J. CHASSAR MOIR.

## HOW TO USE THE MICROSCOPE

*The Intelligent Use of the Microscope.* By C. W. Olliver, A.M.I.E.E., F.R.M.S., M.R.P.S. Second edition. (Pp. 192; 59 figures. 15s.) London: Chapman and Hall. 1951.

This is an excellent introductory handbook for beginners, and the author, with every justification, claims that it combines thoroughness with order and simplicity. He has been at pains to strike the happy mean between "too much theory divorced from its practical implications and the rule-of-thumb type of instruction which remains unexplained." The basic theory underlying magnification, numerical aperture, resolution, and the methods of illumination is discussed at length and in simple terms. There is a valuable chapter on the critical illumination required for photomicrography.

In this edition a special chapter on phase contrast has been included. This method of illumination has entirely done away with some of the most awkward limitations of the microscope because of its capacity clearly to reveal remarkably fine detail in the almost transparent and unstained living cell. The apparatus required for the technique is neither elaborate nor very costly, and the general optical principles underlying this notable advance in microscopical science are clearly explained.

GEOFFREY HADFIELD.

## PRAYERS IN HOSPITAL

*A Collection of Prayers for Use in Hospitals.* (Pp. 218. 4,000 copies free of cost except for postage and packing.*) The General Hospital, Nottingham.

From the General Hospital, Nottingham, comes this book of collected prayers. In addition to the many inspired and varied prayers, this small book sets out, in modified form, suggestions for a set service for each day of the calendar month. Wisely, much has been left to the individual choice of our hospitals. Certain hymns and certain psalms are suggested, certain prayers listed day by day, and the choice is a wide one.

There are prayers for use on special days, there are prayers for cheerfulness, for serenity of mind, prayers too for our hospital staff. Each day has its prayer for thanksgiving, but here let it be noted that, while it is desirable that our patients should give thanks to God, it is asking rather much for the seriously ill patient to give thanks for "health and strength" as worded in the first prayer of the book. Otherwise the prayers are comprehensive, helpful, and imbued with sincere simplicity.

Surely to all our hospitals this book should prove of infinite value. Where daily prayers are customary in the wards, the sister in charge would find this collection of prayers of great help. Alternatively, the service, wherever possible, could be broadcast from a central point in the hospital, reaching every patient at the same time and uniting them in an act of worship.

To many patients may come the wish to study the book at other times, thus drawing to themselves comfort and inspiration during days of enforced inactivity. Indeed, to all patients this book must bring in some degree that spirit of healing which is of such immeasurable importance in helping the work of doctors and nurses.

H. GUY DAIN.

*Obtainable from Messrs. Derry and Sons Ltd., Canal Street, Nottingham, at the following rates for postage and packing: 1 copy 1s.; 2 copies 1s. 3d.; 3 copies 1s. 6d.; 4 copies 1s. 7d.; 6 copies 1s. 9d.; and 12 copies 2s. 4d.

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## CORONARY DISEASE IN DOCTORS

With the present decline in the importance of many of the infectious diseases of childhood—at least as causes of mortality—and with, concurrently, an ageing population, the attention of the epidemiologist is likely to turn increasingly to the ills besetting the middle-aged and old. In the modern sense of the word there is, of course, no difficulty in bringing such diseases into the "epidemiological" net. When taken to comprise always the *group* as opposed to the *individual* aspects of a disease, an epidemiological picture can be drawn of every sickness to which mankind is exposed, just as a clinical picture can be drawn. In diseases of an unknown aetiology such a picture, revealing perhaps an incidence varying in space or in time, in the age of attack or in the occupations of the victims, may well provide a vital clue. The variations, for instance, in the incidence of cancer of certain sites in specific occupations—a group characteristic of the disease—have repeatedly drawn attention to extrinsic factors underlying the production of neoplasms. So far, however, much less has been done to put into an epidemiological setting the other predominant cause of adult mortality to-day—namely, heart disease in its various manifestations. Work in this field will be rewarding, and, to judge by the article in the opening pages of this *Journal*, results may be surprising: the authors have made the startling observation that the incidence of coronary disease in 1947–50 among general practitioners was almost twice as high as the figure for all the rest of the profession—a finding which bears out in a remarkable way the statement in the Spens report about the arduousness of the general practitioner's life.

Dr. J. N. Morris, Mr. J. A. Heady, and Mr. R. G. Barley, the authors of this statistical "thriller," have taken as their basic data certain records of the Medical Sickness, Annuity and Life Assurance Society, Ltd., a mutual benefit organization which has several thousand doctors on its books. Confining their attention to males of ages 35–64 (since most policies terminate at 60 or 65 and few women belong to the Society), they made it their first task to enumerate the doctors of different ages exposed to risk in the four years 1947–50, and then to relate to these age groups the number of first attacks of coronary heart disease which were recorded in the same four years. These attacks are shown in the claims to benefit which were presented either for a non-fatal attack which caused an absence from work of at least a week or for a fatal attack irrespective of duration. The incidence rate thus computed reveals its expected steep rise with age from an almost negligible figure under age 40 to one of 12 cases per 1,000 doctors living at ages 55–59 and to almost 17 per 1,000 per annum at ages 60–64—or approximately, in this experience, one first attack a year in each 60 men in this age group.

Turning from attack to death, the authors found that coronary heart disease was in 1947–50 the largest contributor to the mortality amongst this group of men. After 35 years of age about one-third of the total deaths were ascribed to it. The actual death rate rises from approximately one per 1,000 for men in their forties to 7.4 per 1,000 at ages 60–64, figures not very different, it seems, from those that have been published for medical practitioners in the U.S.A. A particularly notable, if not unexpected, feature is the rapidity of death. Of the 48 fatalities here recorded, 29 took place in the first six days of the first attack and another 9 in the ensuing weeks of the first month—in other words nearly 80% of those dying did so in the first month. Therefore, as the authors point out, the greatest need for therapy is in the first few days of the first clinical attack. Such figures also emphasize the great difficulties which must be overcome if an adequate and convincing trial of any such therapy is to be made—for example, of an anticoagulant. The patients reaching hospital are only the fortunate survivors, in whom the prognosis at that stage may not be very unfavourable: it must at the very least differ profoundly from the initial prognosis. A therapeutic trial to be of any real value will call for the collaboration of the general practitioner. It is of no value merely to be "in at the death."

From these records further light could be thrown upon this question of prognosis by extending the period of observation to cover the decade 1940 to 1950. The history, up to age 65, of all cases occurring in that period of time could be extracted and utilized for this purpose, even though in the earlier years the numbers of doctors at risk was unknown. The authors found 192 such cases—that is, first diagnoses of "coronary thrombosis" in men aged 40–64; and, since division by age suggests no very material changes, it is sufficient to take them as one group. Of these 192 men, 30% died in the first six days and

another 8% in the rest of the first month. Only a further 2% died in the remaining eleven months of the first year. Subsequent fatalities were at the rate of 6% in the second and third years, 3% in the fourth and fifth years, 3% in the sixth and seventh. In short, roughly half the patients had survived three years. Of these survivors of a first attack, about one-third had a second absence from work because of coronary heart disease within the ensuing two years, and about half were attacked again within six years—or, more cheerfully, "about half the men who survived their first illness and returned to work did not have as much as a week's further sick absence for coronary heart disease in the six years following."

With angina pectoris, of which there were 42 first attacks, the outlook is very different. Only 11% died within the ensuing five years, and this is the same order of mortality as is shown by those doctors with coronary thrombosis who had had the good fortune to survive the first month.

Other figures of great epidemiological interest can be derived from the basic rates already quoted of incidence and death at different ages. Using life-table methods—in other words, *presuming that the present rates of incidence here revealed will continue unchanged*—Morris and his colleagues estimate that of 1,000 practitioners aged 35 to 45 some 20 to 25 will have a clinical attack of coronary heart disease before age 50, almost another 40 will be attacked between ages 50 and 55, another 60 between ages 55 and 60, and a further 70 between ages 60 and 65. In other words, "medical practitioners under 45 years of age who have not so far been attacked by coronary heart disease have approximately a 20% chance of being attacked before they reach 65." Similar estimates for the chance of dying give some 70 deaths by age 65 in each thousand doctors setting out from between ages 35 and 45. Though the foundation of these calculations is, admittedly, somewhat slender, it is sufficiently large and sufficiently precise for these figures to give rise to sobering reflection. With events on this scale, "epidemic" certainly does not appear to be a complete misnomer.

How, though, it will be asked, do "doctors" compare with other professional and occupational classes, and how, since their own jobs vary very widely, do they compare with one another? Neither question is fully answered in the present study, but some distinctly intriguing, if interim, figures are presented. It is quite commonly believed that doctors suffer more from coronary heart disease than the rest of the population, but very little convincing evidence has been forthcoming. Impressions based upon the number of doctors seen by an individual or in a clinic are apt to be inaccurate, and probably, as Morris and his colleagues observe a trifle acidly, "the least reliable impressions are those of distinguished physicians who may have a quite disproportionate number of doctors in their personal practice." Failing to observe the attractions of their own distinction, they make the wrong generalization. Their deductions may, however, in spite of logical imperfections, yet prove to be right, since the present figures suggest a relatively high incidence in general practitioners though not in the other sections of the medical profession.

In this latter contrast the authors are on definitely safer ground, since all the records come from the one source—the particular assurance society. This comparison, which gives some very unexpected results, was reached by a series of steps, fully described in the report, which were devised with much care and skill to ensure both accuracy and impartiality in the construction of the required rates. Having found, as already remarked, that in 1949–50 the incidence of coronary heart disease upon general practitioners was almost twice as high as the figure for all the rest of the profession and that the other subdivisions within the latter revealed no material contrasts, they sought, critically and with admirable scientific rectitude, for some flaw in their data or in their treatment of it. They failed to detect any red herring. Extension of the figures to cover the years 1947 to 1950, enlargement of the sample of doctors upon whom the occupational distribution was based, an independent check of those occupations—all left the relative incidence rates virtually unchanged. At ages 40–64 the standardized rate per 1,000 was 8.8 cases per annum in general practitioners and 4.4 in other doctors, and this excess was apparent in each age group.

That being so, is this unfavourable position of the general practitioner in 1947–50 a modern or an old-established characteristic of the "herd"? In answering that question the authors were faced with grave difficulties. The age and occupational structure of the population "at risk" in the war years, 1940–6, was not available, and it had to be assumed that it was the same as had been found for 1947–50, but of course in men some years younger. In defence of this assumption they state that the membership of the Society is a very stable one. The statistical calculations based upon it lead to the observation that there was little, if any, excess coronary heart disease among general practitioners during the war years, but that there was an increase of cases among them in 1947–50 which was not paralleled in the experience of other doctors. This increase does not appear to be due

merely to improving diagnosis in the one group compared with the other, since fatal and non-fatal attacks have equally increased. Beyond those tentatively drawn conclusions the authors cannot, and wisely do not, attempt to go. They emphasize instead the importance of further work, and its extension, beyond the records of the particular assurance society to which their study was confined, to the whole medical profession. It should be profitable, they suggest, not only to explore the antecedents of coronary heart disease among doctors and to expand the clinical picture of it, but also, through the field survey approach, to investigate the possible associations and relationships of the disease. For "Physician, heal thyself" we must perhaps substitute to-day the less euphonious "Physicians, epidemiologically survey yourselves." Dr. Morris and his colleagues have made a good start, and it is to be hoped that with the co-operation of the profession the Medical Research Council's Social Medicine Research Unit will be able to expand its work and carry out systematic studies on a large scale, with the aim of preventing a disease which numbers so many doctors among its victims.

## SUPERINFECTIONS DURING ANTIBIOTIC TREATMENT

The term "superinfection" has come into use to describe fresh complicating infections arising during antibiotic therapy. An initial response to treatment marks the overcoming of the original infection, to be succeeded by a worsening of the patient's condition, accompanied by the appearance of bacteria resistant to the drug which were not found before. These bacteria may be presumed to have been present in small numbers before treatment began and to have found an opportunity to multiply in the circumstances so produced. The obvious circumstance resulting from antibiotic treatment, apart from action on any specific pathogen, is the suppression of the general flora of certain areas of the body. Penicillin exerts this suppressive action mainly in the mouth, owing to its excretion in the saliva, and it is well recognized that in patients receiving heavy doses of this drug streptococci and other bacteria in the mouth are largely replaced by coliform bacilli. Superinfections during penicillin treatment are for the same reason chiefly liable to involve the air passages. The newer antibiotics affect in this way not merely the mouth but the entire alimentary tract, since they are not only administered by this route but also incompletely absorbed. From studies such as those of Bierman and Jawetz[1] it is known that in a patient taking "aureomycin" (chloramphenicol has a less pronounced effect) most of the normal intestinal flora are suppressed for as long as treatment continues, but to some extent replaced by *Pseudomonas*, yeasts, and staphylococci resistant to the drug.

In view of this much more widespread interference with the normal bacterial population of the body it is not surprising that superinfections occurring during the course of or after treatment with these newer drugs have excited much more attention. They are in fact quite common during prolonged treatment, taking the form of a glossitis or an affection of the anal region caused by *Candida* (*Monilia*) *albicans*. This yeast-like fungus is highly resistant to all known chemotherapeutic agents. It was because such infections may take a more serious form, spreading from the mouth to the bronchi and lungs, that the Council on Pharmacy and Chemistry of the American Medical Association decided last year that a warning statement must be printed on the packages of these drugs in the U.S.A., a somewhat startling procedure to which reference was made at the time in these columns.[2] Our comparative inexperience of such conditions in this country is due simply to the scarcity here of some of the drugs causing them, although minor manifestations such as glossitis have become familiar. What may be an example, and an unusual one, of such a superinfection is reported by Drs. J. D. Abbott, H. V. J. Fernando, K. Gurling, and B. W. Meade on page 523. A patient with post-influenzal bronchopneumonia was found to have *Bact. coli* and *Candida albicans* in her sputum: it is not stated that she had had treatment with penicillin during the preceding three weeks of the illness, although such a finding would lead one to suppose it. Despite treatment with penicillin, streptomycin, and chloramphenicol she died, and was found at necropsy to have cavitating pulmonary aspergillosis, although the presence also of *C. albicans* in the lung cavities was confirmed. It is of course possible that the *Aspergillus* infection existed before antibiotics were given and would in any case have been fatal, but when such a condition makes its appearance during the administration of drugs to which the causative organism is wholly insensitive the suspicion that the treatment was doing positive harm cannot be put aside.

More information is needed about the mechanism whereby resistant bacteria and fungi are enabled to

[1] *J. Lab. clin. Med.*, 1951, **37**, 394.
[2] *British Medical Journal*, 1951, **1**, 1196.
[3] Ibid., 1951, **1**, 205.
[4] *J. Infect. Dis.*, 1949, **85**, 268.
[5] *Arch. Intern. Med.*, 1951, **88**, 729.

proliferate and cause lesions during antibiotic therapy. It may be that the suppression of the other flora of the area either upsets a balance normally holding them in check or simply provides them with more pabulum. On the other hand, there is more than a suspicion of positive stimulation to active growth by the antibiotic itself. There is much evidence, recently reviewed by Garrod[3] in this *Journal*, that sublethal concentrations of antibiotics as well as other noxious agents accelerate the growth of bacteria, particularly of resistant species. Directly bearing on the present problem are the findings of Foley and Winter,[4] who showed that treatment with penicillin increased the mortality from *C. albicans* infection in the chick embryo. Pappenfort and Schnall,[5] who reported *C. albicans* infection in 16 patients treated with aureomycin, also found that this drug stimulates the growth of the organism *in vitro*. On the other hand, its growth was also stimulated by solutions in which aureomycin had been destroyed by heat or alkali, and it was unaffected by a pure preparation intended for parenteral injection. They therefore concluded that an impurity was responsible for this effect. Evidently the full truth of this matter is not yet known. Meanwhile we shall be well advised to remember that, although the products of some species of fungi have revolutionized therapeutics, there are other species causing infections which are among the very few still quite unaffected by any form of chemotherapy.

## PSEUDOCYESIS

The woman who firmly but falsely believes herself pregnant and in whom develop many of the symptoms and some of the signs of pregnancy was known to Hippocrates, while the clinical picture drawn by Sir James Simpson in 1860[1] has never been bettered. The reported cases of this medical curiosity total fewer than 500, but, as Simpson wrote, it is "of far more frequent occurrence than the comparative silence of our text-books on this malady would lead you to infer." This is because most of the cases without very remarkable features are not reported, the patient being merely informed sooner or later that she is not pregnant without any deeper search into the origin of her disorder. After being thus dismissed she may go on to trouble another doctor. Fried and his colleagues[2] have recently reported a series of 27 cases of pseudocyesis in which the women, all but one of whom were childless, were examined for endocrinological and psychiatric as well as gynaecological defects. Spurious pregnancy is generally considered commoner in women near the menopause, but Simpson often found it in younger women; and the majority of Fried's patients were in the middle twenties. These patients possess most of the usual symptoms of pregnancy, but hypomenorrhoea is more common than amenorrhoea. One of the most notable features is the appearance of characteristic signs of breast activity, including secretion, even in nulligravidae, but in none of Fried's cases were the uterine or cervical changes typical of true pregnancy. Yet women with pseudocyesis pass superficial examination for pregnancy with ease, chiefly because the possibility of the condition being spurious is not considered, and once the diagnosis of pregnancy has been made the symptoms flourish. In fact such women have been operated on for supposed ectopic pregnancy and even for placenta praevia.[3] Moreover, the symptoms may continue for years rather than months. Simpson relates that Dupuytren, when asked by a lady who insisted she had been pregnant for 14 years, what course she should follow, answered that as the boy must be tolerably well grown by that time the best thing the lady could do was to swallow a tutor, that his education be not neglected.

Fried regards pseudocyesis as a form of conversion hysteria and remarks on the frequency with which the syndrome follows an emotional strain which the patient believes can best be relieved by a pregnancy. A picture of pseudo-pregnancy can sometimes be induced by the administration of gonadotrophins, alone[4] or in conjunction with oestrogen and progesterone[5]; but this differs from pseudocyesis in that if the psychic factor and predisposition are not present the signs do not become progressive, and there are no complaints of morning sickness, enlargement of the abdomen, and "foetal movements." Fried and his colleagues concluded that the amenorrhoea in pseudocyesis is hyperhormonal, whereas most forms of psychogenic amenorrhoea are hypohormonal. In particular, evidence of progesterone activity in most of the women seemed to point to persistent corpus luteum function. This is not a new idea, for Simpson wrote 90 years earlier, "Perhaps we would find the corpora lutea tending to simulate in their development and growth the corpora lutea of pregnancy." But Fried goes further and propounds a hypothesis that the basic mental conflict which appears best solved by pregnancy affects the pituitary by way of the hypothalamus. The reaction in the anterior pituitary, he conjectures, leads to secretion of the luteotrophic hormone at the expense of follicle-stimulating hormone, and the result is persistent luteinic function in the ovary, which could account for most of the clinical features. Whether this hypothesis is correct or not, it remains a fact that, except in minor degrees of the syndrome, merely telling the patient she is not pregnant is insufficient treatment, for either the woman is unconvinced or, after an interval, further symptoms of pseudocyesis bring her again to the doctor. Fried therefore recommends psychotherapy, in order that the patient may obtain an insight into her condition. The best evidence of cure is the return of normal menstruation, the absence of recurrence, and, best of all, a true pregnancy.

1 *Clinical Lectures on Diseases of Women*, 1872, Edinburgh, pp. 363–93.
2 *J. Amer. med. Ass.*, 1951, **145**, 1329.
3 Bivin, G. D., and Klinger, M. P., *Pseudocyesis*, 1937, Bloomington.
4 Brown, W. E., and Bradbury, J. T., *Amer. J. Obstet. Gynec.*, 1947, **53**, 749.
5 Eichner, E., Goler, G. G., Reed, J., and Gordon, M. B., *ibid.*, 1951, **61**, 253.

## LONDON'S WATER SUPPLY IN WARTIME

A report[1] recently published by the Metropolitan Water Board describes the steps taken to maintain an adequate and wholesome supply of water during the war years. The author is Lieutenant-Colonel E. F. W. Mackenzie, the Board's director of water examination. Two schools of thought existed when defence plans were being prepared before war broke out: one considered that it was unnecessary to take precautions against disasters which might not occur; the other maintained that every practicable precaution should be taken to provide against any contingency. Fortunately for the people of London the second view prevailed, and how amply it was justified is set out clearly in the pages of the report. The emergency plans of the Board were concerned first with the repair of mains damaged or broken by bombing (it took some time to discover that concussion from bombs exploding some distance away could fracture water mains without affecting the ground above them); secondly, with ensuring that laboratory tests could quickly be carried out in order to estimate the purity or otherwise of the supply after damaged mains had been repaired; and, thirdly, with the purification by the quickest possible method of repaired water channels after they had been contaminated by sewage. The value of prechlorination—chlorination before filtration—in such times of emergency was proved beyond any doubt, as will be appreciated by all who read the report. So successful was this procedure that in some places the old river intakes (for instance, at Hampton) were opened, and unstored river water was led to the filters. Steps were taken to protect wells also, though fortunately none of the Board's wells was very seriously affected by bomb damage.

The policy of the Board was that no repaired main should be brought back into service without prior disinfection. The methods of disinfection were remarkably successful. Samples were examined from 807 of the 875 large mains which were fractured by bombs, and of these 801 (99.3%) were negative to *Bact. coli*. Of the six which fell below this standard, five contained fewer than 10 *Bact. coli* in 100 ml. The Board's work in fact was so efficient that even in official quarters some credence was given to a dangerous fallacy—that the non-appearance of epidemics meant that the dangers to the population from polluted water had been exaggerated. The report also describes the measures taken to guard against the contamination of water stored in reservoirs by the dropping of bombs charged with such materials as poisonous gases. Daily testing of the water was continued as a routine throughout the war, and on no occasion was there any evidence that poisonous matter had gained access to it. Apart from its technical interest the report is a tribute to the loyal devotion of the members of the team who performed a vital task under very difficult circumstances during six years of war.

[1] Thirty-fourth Report on the Results of the Bacteriological, Chemical, and Biological Examination of the London Waters for the Years 1939–1946. Published by the Metropolitan Water Board.

## VITAMIN-A POISONING

When some writers are warning us of the calamitous results of over-population in a world short of food, and when vast numbers of people are unable to obtain even the minimum of food necessary for survival, it is slightly ironical to read of syndromes caused by over-dosage with vitamins in children. The increased knowledge of nutrition and the special stresses of war have led to the publication of innumerable books and papers on nutrition, both scientific and popular. Some parents, impressed by doubtful evidence of subclinical deficiencies or perhaps even more by skilful advertising, have apparently been concerned to see that their children, at least, shall be free from these preventable illnesses and have given them large and sometimes enormous doses of vitamin preparations.

Reports[1] of vitamin-A poisoning in animals appeared as early as 1933, but the first observation[2] of harmful effects in a child was not reported until 1944. A recent article[3] lists 17 recorded cases, and it is probable that many others have occurred without being diagnosed or reported. Most of the children received about 250,000 U.S.P. units of vitamin A daily for many months, and one had twice this daily dose for 16 months. All the children were more than 1 year old, perhaps because vitamin-A preparations are not very well absorbed in young infants[4] or because of the relative tolerance to large doses. The symptoms are fairly constant and include irritability, anorexia, loss of hair, pruritus, and painful extremities with tender swellings. Radiographs of the long bones show cortical thickening, especially of the ulnae. Biochemical examination of the blood is generally negative, although in some cases the alkaline phosphatase has been high. The amount of vitamin A in the plasma has been high enough for the condition to be diagnosed in all cases. The symptoms disappear within a very few days after administration of the vitamin is stopped.

The differential diagnosis is difficult, largely because of the rarity of the condition. Leukaemia, scurvy, and congenital syphilis present pictures similar in some ways, but fairly easily distinguished, and vitamin-D poisoning is also similar and may indeed coexist to some extent, since the source of the vitamin intake usually contains large amounts of vitamin D as well as vitamin A. Caffey's syndrome[5] bears most resemblance clinically, but there is no history of excessive dosage with vitamin A and the amount of the vitamin in the plasma is normal. There is usually some degree of fever, and the mandible is always involved in the periostitis. Unfortunately the published case records do not discuss the reasons which prompted the parents of these children to give such enormous doses of the vitamin; these might have thrown some light on the effect and effectiveness of methods of education of the public in medical matters.

[1] von Drigalski, W., *Klin. Wschr.*, 1933, **12**, 308.
[2] Josephs, H. W., *Amer. J. Dis. Child.*, 1944, **67**, 33.
[3] Gribetz, D., Silverman, S. H., and Sobel, A. E., *Pediatrics*, 1951, **7**, 372.
[4] Sobel, A. E., Besman, L., and Kramer, B., *Amer. J. Dis. Child.*, 1949, **77**, 576.
[5] *J. Pediat.*, 1946, **29**, 541.

## *REFRESHER COURSE FOR GENERAL PRACTITIONERS*

# CLINICAL ASSESSMENT OF BACKACHE

BY

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An ordered approach to the subject of backache is difficult to achieve. In spite of the discovery about 16 years ago of the prolapsed disk syndrome we are still left with many cases in which speculation replaces certainty, and in which there is no agreement on pathology, diagnosis, or treatment. No matter how one approaches the possible causes of backache, there remains an indefinite fringe of cases in which no certain diagnosis is possible. This group is characterized by either an absence of clinical findings or a clinical finding of painful muscular spasm for which there are alternative explanations.

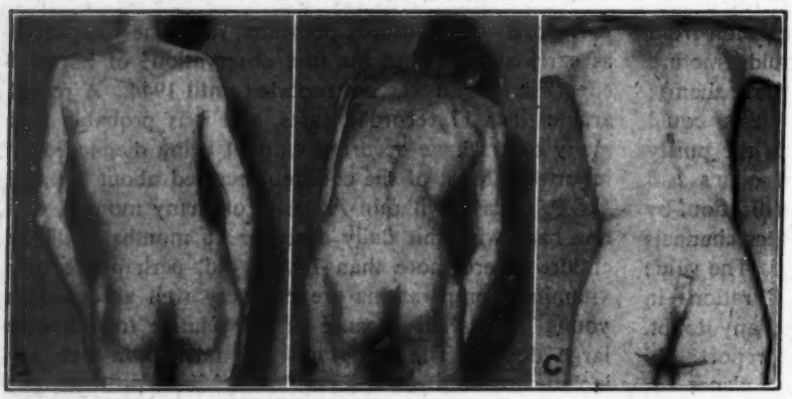

FIG. 1.—*Muscle spasm but no obvious skeletal defect.*—(*a*) A typical sciatic scoliosis showing a lumbar deviation to the left. (*b*) On bending forward the scoliosis usually increases and the patient tends to flex the affected knee. In this case, which is unusual, the scoliosis has altered to the opposite side: this is a recognized occasional variant. (*c*) This shows almost complete obliteration of the scoliosis on lying down, indicating that the primary cause is muscle spasm.

Wherever the seat of the primary disorder may be, it manifests itself to the consciousness in three ways: (1) well-localized pain accompanied by areas of tenderness to pressure—for example, over the spinous processes or a muscular attachment; (2) pain due to pressure on nerve trunks which is felt at a distance over the dermatome supplied by the nerve; and (3) referred pain due to a "spread-over" at higher levels in the nervous system from sources of pain which have no cortical representation—for example, interspinous ligaments, intervertebral disks. A precise anatomical diagnosis can usually be made in the first two groups. In the third group, in which the pain is referred, difficulty arises, and often there is accompanying muscle spasm.

### Muscle Spasm or Structural Deformity?

Muscle spasm usually produces a constant pattern of deformity—a flattening of the lumbar spine on standing, increased tone in one or both erector spinae with limited lumbar flexion, and a scoliosis of the spine increased by flexion. We often find that the patient with muscle spasm is reluctant to bend with both knees straight, but is able to bend with one knee flexed, and that he has a slight reduction in extensibility of the spine. This pattern is most marked in acute protrusion of an intervertebral disk with root pressure. Muscle spasm may or may not be superimposed on a structural deformity of the spine, and the first point in the clinical examination is an attempt to separate defective posture due to spasm from that due to a fixed bony deformity. This can usually be done by careful inspection of the spine while the patient is bending forward, or by noting the obliteration of the deformity when he is lying on the face completely relaxed (Fig. 1).

Fixation of the spine in a *normal* anatomical position, such as may occur in the early stage of spondylitis ankylopoietica, is easily overlooked. Likewise, muscle spasm alone may so fix the spine that there appears to be a structural rigidity. Here it is important to note a few further characteristics of the deformity produced by muscle spasm alone. The spasm is usually unequal, so that forward flexion is disproportionately limited to lateral flexion of the spine and to rotation. When the patient is placed on a couch the spine relaxes and the normal lordosis of the spine reappears or the scoliosis straightens. Such observations exclude the presence of a fixed structural deformity. Unfortunately they do not exclude the presence of spinal abnormality, and in the last resort this can be excluded only by radiography. While this is often necessary, especially in cases with significant clinical features such as recent trauma or a history of pulmonary tuberculosis, wasteful radiography can be avoided only if those cases in which an *x*-ray picture is unlikely to be helpful are excluded.

### Clinical Classification

On the basis of whether muscle spasm and structural deformity are present all cases of backache can be grouped as follows: (1) Obvious skeletal defects and no muscle spasm (Fig. 2)—for example, spondylitis ankylopoietica, healed tuberculosis, and postural deformities. (2) Obvious

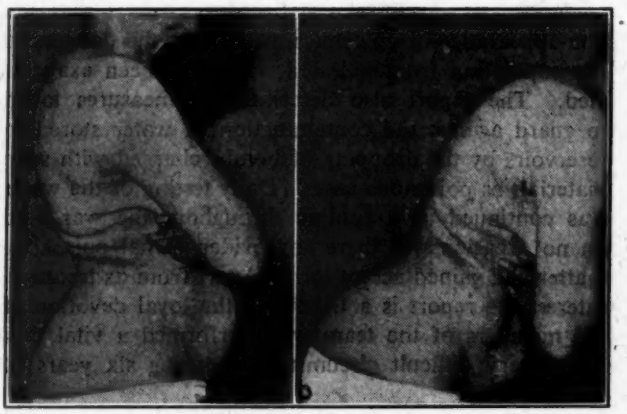

FIG. 2.—*Obvious skeletal defect but no muscle spasm.* (*a*) A marked thoracic kyphosis and lumbar lordosis in an elderly patient unaware of a long-standing spondylitis ankylopoietica. (*b*) On forward bending the curves of the spine remain unchanged. There was an associated gross limitation of lateral flexion and rotation of the spine.

FIG. 3.—*Muscle spasm masking a definite skeletal defect.* (*a*) Flattening of the lumbar spine due to erector spinae spasm without scoliosis. (*b*) Limitation of flexion and persistence of spasm on forward bending. (*c*) Relaxation of the spasm on recumbency, with the appearance of small kyphosis at the lumbo-dorsal junction which was confirmed radiologically to be due to early tuberculosis of the spine with destruction of a single intervertebral disk.

skeletal defects with muscle spasm (Fig. 3)—for example, active tuberculosis, and aggravated osteoarthritis. In this group the deformity is primarily bony and not caused by the spasm. The spasm may serve to show the degree of activity of the underlying disease. (3) No obvious skeletal defect and no muscle spasm—for example, referred pain from visceral causes, poliomyelitis, and hysterical spine. (4) No obvious skeletal defect and muscle spasm (Fig. 1)—for example, lumbago, and prolapsed intervertebral disk. The largest proportion of cases fall into group 4, and to begin with can be safely treated at home, where observation of their reactions to rest in bed will prove a useful guide to the necessity for further investigation.

### Cases with Negative Clinical Findings

The most difficult cases to diagnose may belong to group 3, with negative clinical findings. As clinical examination is so inaccurate in the spine, supplementary radiography is needed. This may result in cases falling into group 1, although the changes in the spine are minimal and only recognizable by radiography. Spondylolisthesis and other developmental abnormalities around the lumbo-sacral junction, early tuberculosis, and localized osteoarthritic changes around a single intervertebral disk are examples of conditions which may be brought to light only by radiography.

Where no assistance is given by radiography the problem may be insoluble, or the answer may be provided only by watching the progress of the disease. It is in this type of case that the history is of primary importance to exclude the visceral causes—for example, renal, gastric, and pelvic disease; to assess the patient's personality in cases following injury in which there may be malingering or hysteria; and to elicit the relationship to pregnancy and the puerperium in the post-natal backache of young women.

### Cases with Muscle Spasm Only ("Lumbago")

The cases in group 4, with muscle spasm but no deformity, are usually classed together as "lumbago." From this group must first be separated those cases due to prolapsed intervertebral disks. This can usually be done by noting the characteristic type of pain: severe aching pain, radiating over a recognized dermatome, often with sciatic pain as well as backache; aggravation of the sciatic pain on coughing, sneezing, or postural changes; complaints of numbness and tingling; and periods of sudden relief, or aggravation.

Examination may show: altered sensation—hyperaesthesia or impaired sensation over a dermatome; absence or diminution of a knee-jerk or ankle-jerk; wasting and loss of tone in the glutei, hamstring, or calf muscles; loss of muscular power in the calf, extensors of the toes, tibialis anterior, and peronei; areas of deep tenderness in the buttock and calf on the affected side; and increased pain on stretching the sciatic nerve (Lasègue's sign and its variations).

Radiological confirmation of changes in the disk space are comforting to the diagnostician, but the essential basis of diagnosis of a prolapsed intervertebral disk remains neurological. Two groups can be recognized: those with objective neurological signs, and the less satisfactory but equally common group with subjective signs only. Where these are characteristic and supported by skeletal signs the diagnosis is definite, but there may be an indeterminate group in which the distinction between acute "lumbago" and prolapsed intervertebral disk remains impossible until the case has been observed for some time, when positive neurological or radiological findings may appear.

After thus excluding prolapsed intervertebral disk as completely as possible there then remains the analysis of the other causes of muscle spasm in the back. These cases form a rather unsatisfactory group. They may be divided up into myofascial strain, paroxysmal "lumbago," and fibrositis or myofibrositis.

*Myofascial Strain.*—The alternative diagnosis to this is a minor disk injury, and this cannot be disproved. Myofascial strain is characterized by a history of strain either by lifting a heavy weight or by lifting in a bent position; a feeling of something snapping in the spine; localized pain on pressure to the side of the spinous processes; recurrence of pain on attempting to straighten from the flexed position; a vague radiation of pain, often bilateral, which never goes below the knee; and recovery with rest and physiotherapy in seven days or less. The diagnosis is seldom confirmed by observation of a haematoma, though there may be an occasional palpable thickening in a muscle. The tenderness is well localized, and complete temporary relief can usually be achieved by procaine infiltration.

*Paroxysmal "Lumbago."*—This may be related to overwork or unusual work. There is seldom any definite injury, and the condition may be recurrent in certain individuals. Repeated examinations often show osteoarthritic changes in the spine in later years of life which are disproportionate to the age of the patient and these may be the basic cause of the attacks. It is characterized by an onset when the patient is flexed and an inability to straighten up; a pain worse on attempting to straighten the back, but the patient can overcome his fears enough to bend forward; a severe pain that has some of the features of cramp, although the individual is not frightened by it and often feels he would like to laugh at himself; an immediate alteration in the quality of the pain after deep massage, with restoration of full or almost full extension; and complete recovery within a week or less.

*Fibrositis or Myofibrositis.*—The onset of the pain in these cases is usually more gradual. The patient may have had similar attacks elsewhere—for example, recurrent stiff neck or fibrositis of the trapezei. It is characterized by slow development sometimes associated with a cold or mild influenzal attack; a tendency for signs to be bilateral—for example, the spasm may involve both erector spinae without flattening of the lumbar spine; areas of tenderness in the muscles that may change from day to day; localized areas of thickening or tenderness over certain recognized areas, notably the twelfth ribs, the lumbo-dorsal fascia, and the iliac crests. Similar tender areas may be found in other muscles at a distance—for example, the trapezei. The recovery is slow, possibly taking two to three weeks, but is complete.

Complete recovery is characteristic of these three groups. A course of rest in bed, local heat, hot baths, and analgesics is therefore not only good treatment but a useful step in confirming the diagnosis. If recovery does not occur within a week a course of more active physiotherapy is helpful. Radiant heat followed by deep massage and extension exercises of the spine will clear up the residual pain in all but a small group of cases.

### Severe Pain

Severe pain in the back may be alarming to the patient and may result in behaviour which causes a suspicion of hysteria. Assessment of the patient's personality is therefore important, remembering that, in general, severe back pain is not a common presenting symptom of hysteria. The following conditions may have to be excluded: acute disk prolapse with severe sciatica; acute prolapse of a disk into the vertebral body without nerve-root pressure; early tuberculous disease, in which the pain is sometimes nocturnal, occurring when the patient relaxes in bed; and collapse of a vertebral body due to any destructive lesion such as a secondary malignant deposit or myeloma. Severe pain unrelieved by rest demands early radiological examination, though rapid recovery with rest is the general rule.

### Conclusion

While it is true that the diagnosis of many cases of backache demands radiography, this examination will be negative in almost half of the cases submitted to it. The simple lines of assessment indicated here will enable a satisfactory clinical diagnosis to be made in the first instance, and this can be modified by observing the patient's reaction to rest in bed and simple comforting domestic measures. It is better to diagnose a case as "lumbago" and treat it as such than to alarm the patient with the current bogy of disk prolapse and to send him to hospital for investigation. In most cases the first line of investigation in hospital will take the form of observing the patient's reaction to rest in bed.

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**Next Refresher Course Article.**—"Food-poisoning," by Dr. Joan Taylor.

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The Department of Health for Scotland recently issued a circular to local authorities containing suggestions for improving the welfare arrangements for old people. Many of the points made may seem rather obvious, but they have not hitherto been summarized in this way. Helpful suggestions are included on how to meet the needs of old people living at home. Briefly, the circular deals with three main problems: old people suffering from neglect in their own homes, the provision of meals for such people, and the use of hospital and residential accommodation for them. One way of meeting the first problem would be the publication locally of the name of the official to whom such cases should be reported, and those local authorities who have not already done so are advised to keep a list of old people, needing attention, who live at home in the area. Health visitors should be used as part of every welfare scheme for the old. A separate memorandum enclosed with the circular discusses the organization of meals services; these should be provided by voluntary effort supported by local authorities. No good reason is seen for charging less than 9d. for a normal meal, as the National Assistance Board can help anyone receiving meals because of special needs. The memorandum emphasizes that the aim of a meals service is not in any way to discourage relatives living within reasonable distance from fulfilling their normal family duties. Finally, the present statutory position is that responsibility for deciding the admission of an old person to hospital or local authority accommodation rests with the hospital or the local authority. Failing the one, the other must be responsible.

## THE BRAIN AS A MACHINE

### SIR HENRY COHEN'S ROYAL INSTITUTION DISCOURSE

The Friday evening discourse at the Royal Institution on February 29 was given before a distinguished company by Sir HENRY COHEN, who addressed himself to the theme, "The Brain as a Machine."

The history of this problem, he said, dated back to Descartes and his period, the first half of the seventeenth century, when the mechanical laws of the "physical" world were being elucidated. Descartes postulated reflex action as explaining movements in which brain and nerves played an essential part, but he realized that man was more than such an automaton, and he regarded phenomena such as memory, judgment, and decision as properties, not of the brain, but of the "spirit" or psyche. Cartesian dualism conceived the human being as body and mind; the body, which was material, could be known perceptively and obeyed the laws of physics and chemistry, while in contrast the mind was not subject to mechanical laws and was only to be known introspectively by its possessor, or, if known to others, only by behaviour, the inferences from which might be misleading. The brain was regarded as the control exchange through which the mind influenced any part of the body.

The physician, said Sir Henry Cohen, was at the outset prejudiced against this conception. He saw a patient whose memory for recent events was clouded, who was indecisive, slovenly, nervous, depressed, but whose behaviour, after a brain tumour had been removed, returned to normal. He saw a child victim of lethargic encephalitis, who before the illness was well behaved, but afterwards lost moral control and developed aberrations. These disturbances were the result of organic disease of the brain and disappeared when the structural damage was made good.

### The Biological Approach

In the seventeenth century physiological phenomena were explained in terms of levers, pulleys, pumps, and the like, but, although these might be accepted as representing muscular movements and movements involved in circulation, not very much of what the brain did could be explained in that manner. The experimental biologist applied the techniques of physics and chemistry to the living organism. This approach was exemplified by Loeb's tropisms, Pavlov's conditioned reflexes, Watson's behaviourism, and Lashley's work on the localization of the "engram." But the story was far from complete, and the approach by analogy was not without its dangers.

Another approach was to see what machines could accomplish. During the last 10 or 20 years electronic computing machines had been developed and these appeared to possess many supposedly rational properties of mind. Sir Henry gave an astonishing list of what such machines could do. They could learn what they were told, apply instructions when needed, read and remember numbers, add, subtract, multiply, and divide, carry out a long chain of operations one after the other, write out the answers, and—an attribute not entirely human in these days—work unattended. They stored in some form the results of preliminary calculations, a process analogous to memory, and made use of the results at the right time. These machines might be only in their infancy. One saw them handling the routing of telephone calls, translating from one language to another, even making strategic decisions in simplified military operations. The answers they gave were not simply "yes" and "no," but more flexible.

### A Clue to Brain Mechanism

The question was how far these automata afforded a clue to the mechanism of the brain. Were the structure and function of the nervous system, so far as known, consistent with the action of an electronic machine? Recent electro-

physiology had revealed that the brain showed significant electrical changes during its activity. They had only to recall the work of Adrian to realize how beautifully these had been discovered. Electrical changes did take place in the brain, but were, of course, extremely difficult to analyse. The interconnexions of the nerve cells were of almost unimaginable complexity.

It was only right to say that as yet there was very little evidence that the central cortical mechanisms were those of an electronic machine. The evidence was incomplete, and in some respects quite easily controvertible, especially some evidence put forward by enthusiasts; nevertheless, the structure of the cortex was not inconsistent with such a supposition, and indeed by hypothesis a number of mental phenomena—memory and so forth—could be explained in that way. But, assuming that the brain did act like an artifact (in the sense of an artificial construct), what was the relationship of the brain machine to the "mind," the "thinking component" of Descartes? If it were accepted that the brain might act like an artifact, then many characteristics which had been accepted as those of abstract mind must be considered as belonging to the brain—for example, memory, recollection, purposive action, communication, prejudice, preference, the weighing of evidence, the formulation of hypotheses and universals. There were many who were prepared to believe that an artifact could do all the things claimed for it so far as mental processes decided by mathematical symbols were concerned, but that a machine could never be made which would fall in love, or enjoy a wine, or write a sonnet, or cast a vote, with all the irrational conduct that implied. But as more was learned of brain physiology more attributes formerly assigned to mind might be transferred to mechanistic abstractions. Lord Samuel had pictured a chess player cogitating whether to sacrifice a queen, and stretching out his hand to make the move, and said that the physiologist could describe the mechanistic movement but not the process which decided the choice. Yet even while that was spoken a chess machine which would play a human opponent was being completed.

In conclusion Sir Henry Cohen said that he could not hope to present such a thesis without obscurity or dogmatism; this was imposed by limitations of time. But it must be understood that there was still a long way to go in attempting to explain functions regarded as "mental" in terms of "brain" regarded as a machine. It was probable that our abstractions of brain and mind were not appropriate to solve in its modern setting this problem which had for so long been posed. Quite different abstractions were required to resolve the difficulty. Despite recent papers, he doubted whether semantic studies alone would suffice to give the clue. What were needed were not only new words but new ideas—a new "thinking cap"—if a satisfactory answer was to be found to the question: "Is the brain a machine?"

# Reports of Societies

## INSECTICIDES TO-DAY

A symposium on insecticides was held by the Royal Society of Tropical Medicine and Hygiene on February 21 at Manson House, W.1. The President, Sir Neil Hamilton Fairley, was in the chair. Six papers, which were not read at the meeting but had been available to persons interested, formed the basis of the discussion.

### Factors Affecting Success

In the first paper Professor G. MACDONALD described the objectives of residual insecticide campaigns. In the prevention of malaria the degree of insect mortality which must be attained for success varied with the number of mosquitoes, the frequency with which the species concerned bite man, and the duration of the extrinsic cycle. Differences in these factors, and particularly in the man-biting frequency, greatly affected the degree of mortality necessary to secure control of the disease. His paper was illustrated by a nomogram which showed the interaction of the variables discussed. He also discussed briefly the factors involved in the eradication of mosquito species as distinct from eradication of malaria. Finally Professor Macdonald mentioned a few subjects in this field on which further research would be profitable.

Dr. A. B. HADAWAY and Mr. F. BARLOW described some of the physical factors affecting the efficiency of insecticides. To destroy insects protected by forest canopy aerosol particles larger than 100 $\mu$ were useless, as they did not pass the first barrier of leaves. Particles measuring 10–40 $\mu$ were more efficient. If oil solutions were applied to absorbent materials, such as the walls of the mud dwellings so often found in the Tropics, insecticide was lost by penetration. There was also a loss of insecticide when mud was sprayed with aqueous suspensions of wettable powders, though the duration of effectiveness on different muds might vary.

The resting habits of insects had to be considered when preparing insecticidal schemes. Contact with D.D.T. (dichlor-diphenyl-trichlorethane), for example, might repel a mosquito before it had acquired a toxic dose. Though mosquitoes might similarly be stimulated to flight by contact with "gammexane" (gamma isomer of benzene hexachloride), the toxicity of that substance was such that they usually acquired a lethal dose from treated surfaces.

### The Newer Insecticides

Dr. J. R. BUSVINE described the newer insecticides. Of these he stated that D.D.T. analogues and "toxaphene" were unlikely to replace D.D.T., except in special circumstances where the very low toxicity of certain of the D.D.T. analogues was an important consideration. As a residual-film insecticide, "dieldrin" should be excellent, as it combined the persistence of D.D.T. with the high insecticidal power of gammexane. Though these insecticides differed in structure they could be grouped together as chlorinated hydrocarbons, and houseflies which developed resistance to one of them might be found to have also become resistant to others in the group. Among non-chlorinated hydrocarbons, "parathion" (diethyl-*para*-nitrophenyl-thiophosphate), an organic phosphorus compound, was the most promising insecticide, though it was more toxic to man than D.D.T. and less effective against bed bugs. In general, compounds of this type were highly toxic to arthropods and to vertebrates, but not very stable or persistent. They might be useful for some agricultural purposes. "Allethrin," a synthetic compound allied to pyrethrins, could be used in the same way as the natural products; it had a better residual action and was free from resinous impurities.

### Resistance

Dr. C. M. HARRISON, in describing the resistance of insects to insecticides, said that within the last four years D.D.T.-resistant fly populations had developed in many parts of Europe, the Middle East, and America. Such flies might also be resistant to other insecticides of the chlorinated hydrocarbon type. Though morphological differences between D.D.T.-resistant and susceptible flies had been reported, agreement about that was lacking. Physiological differences were, however, more likely; thus resistant flies metabolized D.D.T. into the non-toxic substance D.D.E. (an ethylene derivative), whereas susceptible flies did not. This process would not, however, fully explain resistance, as living resistant flies after exposure to D.D.T. might contain enough D.D.T. to kill normal flies. Resistance was clearly inherited, since D.D.T.-resistant flies gave rise to resistant progeny in the absence of the insecticide; the

mechanism of inheritance of this character, however, was complex. There was no good evidence of resistance having yet developed in anopheline mosquitoes.

For the control of resistant species the prevention of breeding by hygienic measures was recommended rather than the use of many insecticides.

### Toxicity to Man

Dr. J. M. BARNES described the action of some synthetic insecticides on man. A few cases of serious poisoning had resulted from the accidental contamination of food with D.D.T. The symptoms included headache and dizziness. In animals poisoned with D.D.T. or gammexane no histological changes had been detected in the nervous system despite the obvious evidence of severe derangement of function. Necrosis and fatty changes might develop in the liver. "Chlordane," "aldrin," and dieldrin in toxic doses acted as convulsants and also produced liver changes. Phenobarbitone might prevent the convulsions, but it did not detoxicate the insecticides. Aldrin had recently been widely used against locusts in Persia, and despite repeated exposure to oily solutions there had been no human casualties.

Dr. R. C. MUIRHEAD-THOMSON and Professors R. M. GORDON and T. H. DAVEY pleaded for standardization of experimental work on residual insecticides.

### Dangers of Upsetting the Balance of Nature

Professor P. A. BUXTON opened the discussion, in which 22 speakers participated. He traced the development of D.D.T. from its origin in Switzerland, through preliminary experiments by Dr. Busvine at the London School of Hygiene and Tropical Medicine, and in America, to its large-scale production and use late in 1943. D.D.T. and the new insecticides had created a bigger change in medicine than had any chemotherapeutic substance. They had made possible the eradication of anopheline mosquitoes, the control of lice among ignorant people, and the abolition of phlebotemus, simulum, and ornithodorus species in certain areas. The sudden reduction in sickness and mortality thus produced was causing a rapid increase in population, with far-reaching results. The problems this caused would increase in geometric progression as these new generations reached reproductive age.

He then went on to discuss the paper by Professor Macdonald, which attempted to define the proportion of anopheles which must be killed (or kept from man) if control of malaria was to follow. It might be possible to get useful information on the age-structure of mosquito populations by marking mosquitoes with radioactive phosphorus. Of the new insecticides he said that a lethal effect in small doses was not in itself important, but extreme lasting power was very important. He was puzzled whether resistance to insecticides was genetic in origin or acquired. If the former, man had not created it, but had only discovered what had been there from the beginning. He protested against those who concluded from only field experiments that an insect was resistant to insecticides. Such people might be seeking to cover their idleness, or their results might be due to adulterated D.D.T. powder. Laboratory experiments were essential in this connexion. Professor Buxton concluded by pointing out that to standardize experimental field work on insecticides could be dangerous. Though one must control or measure natural variables, if possible, it was necessary to avoid limiting freedom of action in this type of work.

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The appointment of Dr. Gérard Montus, of Marseilles, as deputy director of the W.H.O. regional office for Europe, located temporarily at Geneva, has been announced by Dr. Brock Chisholm. Dr. Montus has been on the staff of the World Health Organization since 1949, serving first as European regional adviser on maternal and child health and later as chief of the social and occupational health section, W.H.O. headquarters.

# Correspondence

*Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.*

### Corneal Grafts

SIR,—The attention of Parliament has been called to consider an alteration in legislation so as to increase the supply of donor eyes by voluntary means for corneal graft surgery. In recent years, thanks to the French revival and improvement of an old method which is mainly free from complications, the scope and safety of corneal grafting have been increased and about 50% of suitable cases treated by this method show restoration of sight to varying degrees. The shortage of donor material is nation-wide and becomes steadily more so since it mainly depends to-day on the use of pathological eyes which have to be removed for disease not affecting the cornea: coincidentally the scope and applications of corneal graft surgery steadily expand.

Mr. B. W. Rycroft, who has been engaged in corneal graft surgery since 1936, has initiated a movement to obtain this alteration in the law and to ensure a legal and voluntary reservoir of adequate donor material. He should have the full support of our profession and he has my entire encouragement also in his endeavours to place British ophthalmology in the forefront of international practice in corneal grafting. It is true that our eye surgeons have been badly hampered hitherto in this type of work by war, lack of special instruments, untrained personnel, and inadequate donor supplies. But only the shortage of donors now restricts the expansion of graft surgery in this country, and it is to be hoped that Parliament will wisely amend the old Anatomy Act to solve this difficulty which obstructs modern eye surgery in the treatment of our fellow countrymen who are blind by corneal disease.—I am, etc.,

Royal College of Surgeons of England, London, W.C.2. CECIL WAKELEY.

### Educating the Public about Cancer

SIR,—Drs. T. A. Watson and O. D. Beresford in their letter on educating the public about cancer (January 12, p. 107) tell of the "intensive publicity" in the U.S.A. and Canada, and more particularly in the province of Saskatchewan, where there has been "a marked increase in the percentage of early cancer seen in the Government clinics." However, that is only part of the story—a part that makes it imperative that more of it be presented. They might have told that, in spite of the publicity, propaganda, heavy financial outlays, and super-organization with increase thereby in the percentage of early cases, there was no reduction and no change in the trend of breast cancer age-specific mortality rates, but that these rates maintained fair levels, as they did, too, in each of the other provinces, some of which had no cancer control programme and no evident increase in the proportion of early cases receiving attention. (The book-keeping of all mortality for each of the provinces was done by the Dominion Bureau of Statistics.) They might have added that the level trends in the Canadian provinces were practically uniform with those in the State of Massachusetts, where there has been a long-established and very active programme of control, and were uniform also with the trends in England, where the complaint has been frequently made that propaganda and promotion for the control of cancer have been less than on this side. They might have concluded that whether or not early treatment can prevent the spread of the lethal metastases and thus prevent death in an occasional case of breast cancer, allowed as a possibility by Sir James Paget in 1853, the evidence to-day fully confirms Paget's view that early treatment cannot prevent death in more than that hypothetical occasional case, and, owing to the nature of breast cancer, cannot materially postpone death where it does not prevent it. (Of course,

treatment can cure non-metastatic non-lethal breast "cancer," indistinguishable morphologically from lethal (metastasizing) breast cancer. However, cure of all such non-lethal lesions—which in consequence of educating the public (and the profession) about cancer have greatly increased among treated cases—would not reduce the cancer death rates.)

They might have told that, in spite of the programme in Saskatchewan and the shift to earlier cases, the age-specific mortality rates for female cancer as a whole for that province fail to show any reduction or any difference in trends from those of other provinces without control programmes. They could have added, too, that there was nothing in the Massachusetts rates any more than in the English rates to suggest that any appreciable control of cancer mortality as a whole had been achieved. They might have concluded that the evidence usually accepted as supporting the faith in early treatment is not unequivocal evidence, but that on the contrary the experience of the past 15 to 25 years shows indubitably that the great advantages claimed for early treatment "just do not exist in practice."

Indeed, the authors might have used the accumulated experience in demonstration of the abiding truth of Farr's words that "death is not deceived by sham defences." And they might well have quoted him further, that "false evidence injures instead of aiding the cause." As Dr. James F. Brailsford has so ably pointed out (November 10, 1951, p. 1154), there is nothing spectacular to offer and what there is had best be done quietly.

The evidence on which this note is based is summarized briefly in the February number of *Surgery, Gynecology, and Obstetrics*.—I am, etc.,

University of Toronto. N. E. MCKINNON.

## Penicillin Cream Dangerous?

SIR,—I read with interest Professor D. M. Dunlop and colleagues' analysis of National Health prescriptions (February 9, p. 292), and noted that 4% of prescriptions were for penicillin.

It is a pity that so much of this expensive drug should be wasted by its use in unsuitable cases and in ineffective doses, but it is still more regrettable that 40% of these prescriptions should be for penicillin cream. It may be that there are a few cases in which penicillin cream is indicated, but they must be very few, and in most cases parenteral penicillin would be far more effective and economical. For the vast majority of skin lesions, ulcers, and wounds penicillin cream is not only useless, but results all too often in the production of a resistance to the drug and, worse still, in severe sensitization.

In clinics for varicose veins and peripheral vascular disorders, where 1,000 or so cases are seen yearly, almost every patient who has had an ulcer, eczema, or infection of a toe from diabetic or other form of vascular deficiency has been given penicillin cream before he comes to the clinic. I can remember only two or three patients who felt the cream had done them any good; almost all discovered for themselves that the cream aggravated the lesion, and a great many developed severe, intractable, persistent, and often generalized skin eruptions.

I am convinced that penicillin cream, like sulphonamide ointment, has done far more harm than good, and it would be a benefit to the public, and to the Treasury, if its sale were prohibited.—I am, etc.,

Liverpool. J. B. OLDHAM.

## What Doctors Prescribe

SIR,—Referring to the article by Professor D. M. Dunlop and colleagues (February 9, p. 292) and subsequent correspondence (February 23, p. 435), there appear to be some points requiring emphasis:

Is there any special virtue in an extemporaneous pill, such as quoted over pil. cascara co. *B.P.C.* 1934? Formulae such as those included in the *B.P.C.* and *N.F.* are usually the result of expert opinion, with rare exceptions such as aspirin mixtures.

With what frequency are the various *N.F.* preparations prescribed? Despite inclusion, there are preparations therein which are only rarely prescribed.

What is the position where a proprietary preparation and an official preparation cost the same? So far as I know the prescription of "sulphadimidine" does not lower the cost, apart from the other examples of insulin, etc., quoted.

The practitioner's right to prescribe must not be questioned, and the basic problem is to educate the doctor (if one may use this word) to the vast number of official preparations available. In this connexion certain points should be borne in mind:

"Academic" titles may be all right, but sometimes these strain the doctor's memory. Perhaps it is in a good cause to use the same ingenuity as the makers of branded goods and produce simple titles. For example, "Easton's syrup" is more easily remembered than the full official name.

When included in the *N.F.*, *B.P.* and *B.P.C.* preparations should also have the formulae (%) included and not just the bare official name.

The *N.F.* should be made more comprehensive, and when a particular proprietary formula becomes extensively used the compilers should consider whether or not to insert a similar formula.

The same effective publicity, at the same cost, would be needed to achieve this as employed by the manufacturers of proprietary products. Merely sending the information to doctors is no guarantee that the matter would be thoroughly digested. Perhaps one of the cheapest ways in the end would be to supply every doctor with a free copy of *The Extra Pharmacopoeia*, when the new edition is published, which offers intriguing reading. Such an act does not, however, overcome the problem of keeping therapeutics up to date. How many doctors know the current view that the soluble sulphonamides, rather than the insoluble, are indicated in the early stages of dysentery?

Finally, before asking doctors to do anything, the Ministries should take care that their own house is in order. From time to time official publications, films, etc., use proprietary names for cotton and gauze tissue, collod. flex., and petroleum jelly.—I am, etc.,

Newcastle-upon-Tyne. A. FORSTER.

SIR,—While acknowledging the great debt we owe to Professor Dunlop and his associates for the very painstaking and illuminating review of prescribing in the first few months of the Health Service (February 9, p. 292) I would like to point out the following two facts which have apparently been overlooked:

1. The form E.C.10A, though approved by the medical profession, has not been agreed to by the retail chemists and has never been implemented.

2. The fact that E.C.10A has never been in existence makes a number of the observations quite valueless, especially over sulphonamides and penicillin.

Their observation that inoculation for colds is not much good is not my experience after 20 years' experiment on myself and dozens of patients who return to me in the autumn year after year for fresh inoculation. The actual inoculation used may be one factor in this sphere.

I would like to add that, with all the statistical material now available quarterly, surely three years is a long time to take over a first instalment of what is obviously a very valuable field of research, both clinical, financial, and political. As many local executive councils have apparently been issued with this report and may be drawing their own conclusions, I feel that, the sooner their attention is drawn to the fact that some of these conclusions are inaccurate and misleading, the better they may be able to adjust their preconceptions.—I am, etc.,

Hadlow, Kent. J. B. MARSHALL.

SIR,—Dr. Frewen Moor (February 23, p. 436) recommends the use of vaccine for the modification or prevention of severe catarrh, as he so graphically describes. My own

experience has been the same as his. If the successful use of vaccine in such cases is not reported, many will be discouraged by the large-scale experiments which have no bearing upon their selective employment.

Obviously where catarrh is caused solely by a virus bacterial vaccine can have no prophylactic value. But what I have noticed is not that vaccine-treated patients escape catarrh altogether—they do not: but they avoid severe and abnormally prolonged attacks such as they had previously been liable to incur, and even mild attacks become less frequent.

This is understandable if the catarrh virus is responsible only for the initial stages of an attack, prolonged exacerbations being caused by a latent bacterial factor coming into action when the virus has prepared the ground.

This latent bacterial infection may in turn keep the local level of resistance low to virus infection. Thus a vaccine which discourages the bacteria would tend to reduce the frequency as well as the severity of attacks.

—I am, etc.,

Leyburn, Yorks. S. G. PEILL.

## Cost of Treatment

SIR,—Your leading article on "Bacterial Resistance to Chemotherapy" (February 23, p. 425) is timely, and the opening sentence that "the clinical exploitation of a new antimicrobic drug passes usually through two main phases, the first being over-enthusiastic and somewhat indiscriminate, and the second more critical and restrained," bears repetition. In the same issue Drs. B. Wolman and A. Holzel (p. 419) describe the rapid response to "terramycin" of 34 young patients with pneumonia and 10 patients with upper respiratory tract infection. I suggest that their paper may illustrate the first phase of terramycin therapy, and before the second phase is reached there must be reports of further investigations backed up by control series.

There are, of course, patients suffering from respiratory infections who derive great benefit from chemotherapeutic agents, but many children respond to routine treatment without antibiotics equally rapidly and well. In a recent consecutive series of 24 children suffering from respiratory infections treated in the wards of the Liverpool University Department of Child Health at Alder Hey Children's Hospital, half the patients were treated with sulphonamide or penicillin, and the others (alternate patients) received no specific chemotherapy. The clinical response was similar in both groups, and in almost all patients the temperature had returned to normal within 24 hours. The time of disappearance of the radiological shadow was the same, and the average length of stay in hospital was 15½ days in each group.

Attention should also be directed to the economic aspect. The approximate cost of treating an infant weighing 15 lb. (6.8 kg.) for a five-day period, using the accepted dosage of chemotherapeutic agent, is 1s. with sulphamezathine, 3s. with crystalline penicillin, 9s. with "distaquaine" fortified penicillin, 4s. 6d. with streptomycin, 23s. 6d. with chloramphenicol, 33s. with chloramphenicol palmitate, and 27s. 9d. with "aureomycin." Terramycin is not generally available in this country at the present time, but its cost will probably not be less than that of aureomycin.

It is obviously right that clinical trials of new preparations should be carried out, but the value of such trials is much increased when one or more control series is also studied.—I am, etc.,

Liverpool. R. McL. TODD.

## Cost of Anaesthetic Techniques

SIR,—While I agree with Dr. F. F. Waddy (February 16, p. 384) about the desirability of economy in the use of anaesthetic gases, I feel it important to emphasize the dangers of using minimal gas flows.

The technique he suggests, using a gas flow of 2 l./min. nitrous oxide and 0.4 l./min. oxygen in conjunction with a carbon dioxide absorber, casually viewed appears reasonable; but, assuming a patient with a minute volume of 5 l. and an oxygen consumption of 200 ml./min., the mixture inhaled will contain less than 11% oxygen, scarcely a fit mixture on which to maintain a patient undergoing radical mastectomy.

Increase the oxygen supply by 0.2 l./min. and the proportions will be atmospheric; but, with such a small margin for error, what machine can be trusted sufficiently for one to dare use the optimum percentage?

I am afraid that the combination of human and mechanical fallibility renders this method too hazardous to the patient, who I am sure would prefer Dr. Bullough's "extravagance."—I am, etc.,

Manchester. C. P. BROWN.

## Hypnotism in Medicine

SIR,—Your recent correspondence on the use of hypnotism for the treatment of asthma has just come to my notice. The following case might be of interest to your readers.

An unmarried charwoman of 49 suffered from asthma since the age of 13. Recently the attacks had been growing more frequent, each attack being of longer duration than the preceding. Two months ago she passed into status asthmaticus, which she had done on previous occasions. This time, however, the attack refused to yield to orthodox—i.e., adrenaline and aminophylline—therapy in the ward of a large general hospital. After remaining in this condition for 10 days it was feared that cardiac failure might supervene, and she was referred to me.

The patient was hypnotized and told that the asthmatic attack would stop immediately; within a few seconds the rhonchi, which could be heard with ease many yards away, disappeared and the patient's comfort and colour improved. After five minutes of repeated suggestion that the wheezing would go and expiration would become less difficult the attack had completely terminated.

It is of interest to learn that up to the time of writing the patient is free of attacks and has been discharged from the hospital. Previously she had had at least one attack per day for the last five years.

It would seem that, when all other methods of treatment fail, before resorting to cortisone an attempt at relief by hypnotic suggestion might be made. The least that can be claimed for it is that it is a great deal cheaper.

For those concerned with the psychological aspect of asthma the history of the case is of interest. The onset of this patient's asthma coincided with her removal with her seven sisters from their home to a convent. The mother was permanently deprived of the custody of the children and the father sent to prison for incestuous relationship with his daughters, the patient included. The ages of the girls ranged from 10 to 19, and they had been submitted to this relationship since the age of about 5. One other sister developed asthma at precisely the same time as the patient.—I am, etc.,

East Grinstead. A. A. MASON.

## L-*Nor*adrenaline as Vasoconstrictor

SIR,—The use of L-*nor*adrenaline for the treatment of hypotensive states has recently received considerable attention (Dr. H. C. Churchill-Davidson, December 29, 1951, p. 1551). Dr. R. Ghose (February 16, p. 385) recommends intravenous infusions lasting up to 48 hours. Although our experimental work is incomplete our recent findings compel us to draw attention to certain possible dangers in these procedures.

In man intravenous adrenaline increases liver blood flow, *nor*adrenaline decreases it (Bearn *et al.*, *J. Physiol.*, 1951, **115**, 430). Our own experiments in animals have shown that in a long-continued infusion the dilator action of adrenaline (which is in fact reflex in nature, a response to the raised blood pressure) is superseded by a progressive diminution in blood flow. This effect is even more marked in the case of *nor*adrenaline, the direct action of both drugs on the liver blood vessels being thus vasoconstrictor. Moreover, our evidence demonstrates that *nor*adrenaline and adrenaline both have a marked constrictor effect on the terminals of the hepatic artery, a point of significance, as the main oxygen supply of the liver is through these channels.

There are important implications in these observations. The organs normally used by nature in the regulation of peripheral

resistance are structures such as the skin and bowel, whose vascular needs may be important but are rarely urgent. Our observations indicate that liver blood flow is a function aloof from such petty adjustments. Changes in blood pressure produce only transitory effects, and surprisingly high levels of flow are maintained even in the presence of low systemic blood pressures. The primary object of blood-pressure regulation is, after all, to maintain an adequate blood flow through vital organs. There are few organs more vital than the liver. There are few organs more readily damaged by an inadequate supply of oxygenated arterial blood (E. J. Delorme, *Lancet*, 1951, **1**, 259). To preserve blood pressure by subjecting the liver to long periods of relative ischaemia would seem to be a hazardous and unphysiological procedure—particularly if it has already been exposed to possible harm from varying periods of hypotension.

It might not have been thought necessary to bring these points forward were it not already known that long periods of adrenaline infusion result in severe and irreversible liver damage (F. A. Bainbridge and J. N. Trevan, *British Medical Journal*, 1917, **1**, 381). The evidence we have mentioned shows that with respect to the liver *nor*adrenaline has vascular effects similar to, though even greater than, those of adrenaline. There is no suggestion that brief infusions might be harmful, but it is our view that *nor*adrenaline administration for periods longer than an hour or so is a potentially dangerous procedure which might merit further investigation before its general adoption.—We are, etc.,

Departments of Physiology and Pathology, University of Bristol.

J. GRAYSON.
D. H. JOHNSON.

SIR,—Dr. H. C. Churchill-Davidson (December 29, 1951, p. 1551) mentions the use of L-*nor*adrenaline for the treatment of collapse under spinal analgesia. This drug has been found most useful in the prevention—rather than the treatment—of blood-pressure falls under spinal analgesia. With a few modifications the spinal technique described by R. Marcus and T. C. Gray (*Brit. J. Anaesth.*, 1949, **21**, 182) has been used in this centre for some time for Wilson Hey prostatectomy, the blood pressure being maintained at the pre-operative level by 1:250,000 adrenaline drip. In recent months L-*nor*adrenaline has been substituted for the adrenaline. The series is not yet big enough to make a statistical evaluation of this innovation, but the following clinical impressions have been formed of the differences between L-*nor*adrenaline and adrenaline.

With L-*nor*adrenaline much less solution is required to maintain blood pressure. In several cases when only 10–20 drops per minute were required it was considered advisable to change to 1:500,000 solution in order to give adequate control of the pressure.

The drip can be discontinued sooner with L-*nor*adrenaline than with adrenaline without any fall in blood pressure. With the latter it was not unusual to find that the infusion had to be continued for 12 or more hours. In 65% of the cases in which L-*nor*adrenaline was used the drip was discontinued in the theatre, and in the remainder the longest time it was required to be continued in the ward was four hours.

The tachycardia of adrenaline was replaced by bradycardia. Pulse rates observed were mostly between 55 and 68 per minute. Blood-pressure changes do not occur so quickly with L-*nor*adrenaline as with adrenaline.

Despite the theoretical objection that L-*nor*adrenaline produces vasoconstriction of the renal vessels (H. C. Churchill-Davidson and others, *Lancet*, 1951, **2**, 803) no cases of post-operative anuria have occurred. The convalescence of this series of cases has not differed from those in whom adrenaline was used. In a few cases E.C.G. tracings were taken before, during, and after the use of L-*nor*adrenaline and no abnormalities have been noted.

On one point the findings are at variance with those of Churchill-Davidson—namely, the diastolic pressure was not always maintained as well as the systolic. In about half the cases there has been an increase of 5–15 mm. mercury in the pulse pressure, but in the remainder this was unaltered or slightly decreased.—I am, etc.,

Liverpool. JOHN W. DUNDEE.

## Frequency of Micturition

SIR,—Mr. H. P. Winsbury-White in his article on this subject (February 16, p. 372) makes special reference to children and to enuresis in which, as we all know, a frequency but less commonly an urgency of micturition occurs. His remarks upon cerebral impulses, emotional disturbances, and the effect of profound sleep are readily conceded, but his main theme is upon those chronic inflammatory processes which can occur in the posterior urethra and upon other physically abnormal findings.

The question which perturbs me is whether or not one should, as a necessary part of the physical examination of these children, submit them to a routine urethroscopy and cystoscopy. A long experience of enuresis in consulting and clinic practice, and in the taking of some of these cases into my private home where they have been watched by day and by night, suggests that one is not justified in doing this unless all methods of approach to the child and his home, together with every possible attack upon the condition itself, have met with absolute failure in which there is no improvement of any kind, however transitory. Such matters as residual urine, meatitis, adherent prepuce, vulvitis, and cystitis can be readily recognized, though their successful treatment does not necessarily, and in my opinion not usually, bring relief to the enuresis. Tonsillectomy has at times "cured" enuresis, as has circumcision, but this is not because the child has lost a conceivably diseased or unduly hypertrophied tonsil or a long and tight foreskin; such conditions are more rightly regarded as coincident and not causal, and this probably holds good in the rare instance of spina bifida occulta. But the presence of granulomata or polypi in the posterior urethra, or of a localized or generalized urethral contraction, is of a very different order and might well be regarded as causal, and all the psychotherapy in the world will be as so much wasted time and effort if the child is all the while suffering from something of this kind which, tragically, has been missed because it just was not considered in diagnosis.

It may well be that some of that hard core—as it has been called—of enuretic children who show no response to any form of treatment are suffering from the conditions which Mr. Winsbury-White has described; and, if this is so, his article can scarcely be overestimated in value.—I am, etc.,

London, W.1. MAURICE L. YOUNG.

## Prostatic Obstruction and Inguinal Hernia

SIR,—Of the many benefits conferred on the elderly patient by modern closed methods of prostatectomy not the least is the great reduction in the incidence of post-operative incisional herniae. In contrast to this was the bulge which developed at the site of the tube track in nearly 50% of the patients after open prostatectomy and necessitated the wearing of a belt, often in addition to the apparatus already worn to control an inguinal hernia.

In many patients divarication of the recti will follow a vertical incision of the linea alba whether the bladder has been drained or not, and this is always a source of increasing weakness to the abdominal wall. For this reason a transverse suprapubic incision as recommended by Mr. Thomas Moore (February 16, p. 362) is becoming increasingly popular among urologists, and the supra-symphysial Pfannenstiel scar is now no longer to be regarded as the exclusive property of showgirls and artists' models. Ever since I was converted to this incision some years ago I have not ceased to marvel at its efficacy—in fact, the thin and flabby type of abdominal wall sometimes appears to have been even strengthened, and on more than one occasion I have observed the disappearance of a small direct bulge subsequent to operation.

Mr. Thomas Moore's concern over the problem of an obstructing prostate complicated by an inguinal hernia is shared by all urologists, and recent reports both in this country and in America make it clear that it is becoming an increasingly common practice for both conditions to be dealt with simultaneously by open operation whenever conditions permit. Personally I believe that simple removal of an indirect hernial sac is all that should be attempted in these cases, and for this purpose the abdominal extraperitoneal approach to the inguinal (or femoral) canal as described by Henry (*Lancet*, 1936, **1**, 531) is ideal. Removal of the sac by this method is usually surprisingly easy, but when it is adherent gentle traction on the testicle in the scrotum by an assistant, as recommended by Henry, facilitates the dissection. After removal of the sac a patulous internal ring should be

narrowed by an occluding suture to prevent recurrence, care being taken not to damage the vas as it enters the internal ring postero-mesially or the internal spermatic vessels postero-laterally. Irreducible herniae, of course, can be dealt with only through a separate scrotal incision, and in these cases a very adherent sac can be left *in situ* after its contents have been freed and returned to the abdomen, the neck of the sac being occluded by a ligature from the abdominal aspect.

I believe that the extra time spent on an elaborate repair of the inguinal canal, especially when the hernia is a direct one, is quite unjustifiable in this class of patient, and furthermore, even though the dangers of post-operative extravasation of urine are admittedly slight with modern closed methods, the development of a wound infection in this area from such a cause would be a serious complication, and one which might well develop into a major disaster.—I am, etc.,

London, W.1. JOHN SANDREY.

SIR,—I read Mr. Thomas Moore's article on prostatectomy and herniorrhaphy (February 16, p. 362) with interest, as for some time I have been using a similar procedure. Though the end-result is much the same, the method is different, and I think, therefore, it may be worth while describing it. Since Professor A. K. Henry first described his method of extra-peritoneal approach in the repair of femoral hernia I have used it almost exclusively for all types of femoral hernia though not for inguinal hernia, although it had been described by Cheatle many years before for inguinal hernia.

Of recent years, using a transverse incision for prostatectomy—almost always the vesico-capsular (Ogier Ward) operation—I have also removed any hernial sacs extra-peritoneally. After the prostatectomy is completed my assistant retracts the rectus on the side of the hernia and by a process of traction on the peritoneum and gauze dissection the neck of the sac comes into view. Once the neck is isolated it only requires a little patience and gentle blind dissection by a finger in the canal to produce the entire sac. This is then divided at its junction with the peritoneum of the abdominal cavity and the opening either ligated or sutured like any other opening in the peritoneum. I have not found it necessary to do any repair to the canal.

Whether or not I have removed a hernial sac, it is my practice also to ligate the vas where it passes over the pelvic brim, as I have found that occasionally there is a little reaction and discomfort in the wound when the vas is approached and ligated through the usual separate incision near the root of the scrotum.

—I am, etc.,

Hartlepool. H. M. GRANT.

## Experiment in Peacemaking

SIR,—It is generally accepted that when a situation is desperate anybody, however obscure, is justified in submitting a plan of action—provided always that he has something original to propose and that it is the outcome of hard thinking. It was strictly on that basis that I proposed (*Journal*, December 1, 1951, p. 1337) that our Association should undertake an experiment in peacemaking. I would be grateful if I might be allowed to outline the argument on which my proposal was based.

It seemed to me that the threat of another world war concerned the medical profession in two ways: the state of the public mind, and our responsibilities in the matter of the prevention of war. I felt that it was high time that the problem was tackled as a scientific problem—and very largely a medical problem—in which research and experiment were more than usually important; that consultation and co-operation with other professions were required, and that it was essential that this problem should be dealt with quite regardless of political, or even religious, opinions.

First there is the fact that the public have suffered so much from the consequences and the threat of war that they are sick in mind and sick at heart. It is not possible in a letter to do more than indicate what troubles them. An example of what bewilders them is a policy which does away with all the air-raid shelters and at the very same time talks of plans to deal with a million casualties in one place in one night. Next, they are horrified by the ever-increasing cruelty and ruthlessness of never-ending war. They are tormented by questions such as what would happen if an enemy were to announce that all captured airmen would be tortured to death—not at all an unlikely announcement. Another anxiety is the extension of the policy of "frightfulness." It gets worse and worse and seems to be taken for granted. Take, for example, the problem of enlisting women in the Forces. In the first world war all the most influential women took up nursing, but in the second world war nearly all of them volunteered for the Forces. These are examples of questions that are weighing on the public mind, and there is great danger that morale may be worn by anxiety and hope deferred down to breaking point.

For this reason, if for no other, we must face the question of our responsibility in the prevention of war. I would not suggest for a moment that the B.M.A. should take any part in politics: what I do suggest is that we have no right to say that the prevention of war is a matter for the Government and no concern of ours. The right view, I am sure, is that it is extremely unlikely that any government can prevent war unless organizations such as the B.M.A. do take their part in making peace in their own field.

I would suggest that a reasonable view of the Russian problem is this: The foreign policy of the Kremlin is extremely obscure, one of the great difficulties being that so many of their speeches are almost unintelligible. The great fear is that they may be scheming to make the whole world Communist, if necessary by force—perhaps with the idea that this is the only way to prevent wars. However that may be, it is obvious enough that the present Russian Government is almost impossible to deal with. Negotiations drag on interminably, and the bills for armaments keep mounting up. We may be bankrupt long before we reach any settlement with the Kremlin.

But if we turn from the Russian Government to the Russian people the outlook is very different. There does not seem to be any difficulty in understanding the ordinary people; they seem to be very much like ourselves and quite different from the Nazis. There is nothing to show that they want to make the whole world Communist, certainly not by force. All the evidence is that they are very willing to live and let live. There seems to be no reason at all why the ordinary people on the two sides, if they could get to know each other, should not make friends. It is clear enough that in the end the only way to prevent war is to make peace day by day, year by year, and my suggestion is that that is the province not of the Government but of the people themselves. I would say that it is the function of the Government to defend the peace until the ordinary people have made it clear that armaments are no longer needed. The cold war, with all its hideous risks, will go on until the ordinary people make peace.

Our concern is that the ordinary man in the street can do nothing at all on his own, but in these days people are organized in all sorts of ways and can get into touch with their opposite numbers in other countries through their professional, business, and trade associations. And that is where our own responsibility lies. Just as each of us has a dual responsibility, as a doctor and as a citizen, the responsibilities of the B.M.A. extend beyond the field of pure medicine in this country. Nearly a hundred years ago Sir James Paget addressed the students of St. Bartholomew's Hospital on two or three occasions on the subject of their responsibilities. He was one of an illustrious line and he voiced a tradition going back in one hospital for nearly a thousand years. It is impossible to believe that what he said can apply only to our own patients or our own country. His teaching was that our responsibilities "are as various as are the ills that flesh is heir to; they are as deep as the earnestness with which men long to be delivered from suffering, or from the grasp of death." We are bound "by the weightiest responsibility to God and man. . . ."

—I am, etc.,

Wetherby, Yorks. R. L. KITCHING.

## Debauchery of Honest Words

SIR,—Kindly allow me to express a strong protest against the admission to the *British Medical Journal* of the phrase "spectrum of antibacterial activity" in the summary of the article on "terramycin" by Drs. B. Wolman and A. Holzel (February 23, p. 419). The word "spectrum" means an image—usually that formed by rays of light—in which the parts are arranged in a progressive series according to their refrangibility (*O.E.D.*), and how an image can have antibacterial activity should be explained by those who perpetrate the usage I complain of.

It was once said of the word "allergy" that it had become so debauched by misuse that it would be fortunate if it could be dropped from the vocabulary of scientific

language, and now it is the turn of "spectrum" to be ill-treated.

I ask for your powerful editorial aid against this debauchery of honest words.—I am, etc.,

Belfast. H. G. CALWELL.

### Appeal for Specimens

SIR,—The Medical Research Council's Dental Research Unit is in urgent need of certain post-mortem material for its investigations into dental and associated disease. In particular, the jaws, or parts of jaws, with teeth *in situ*, are required from healthy mouths; from cases with gingivitis but no other specific lesions; from cases of ulcerative stomatitis; and from epileptics formerly under "epanutin" (sodium diphenyl hydantoinate) control. When any of this material is or is likely to become available it is requested that this unit be notified (Telephone: Brixton 4744). Arrangements will then be made to collect the specimens, which if possible should be removed and fixed in 10% formalin soon after death.—I am, etc.,

Medical Research Council,
Dental Research Unit,
King's College Hospital Medical School,
Denmark Hill, London, S.E.5.

J. J. D. KING,
Director of Unit.

## POINTS FROM LETTERS

### Overwhelming Response

Miss D. J. COLLIHOLE (Director, Periodicals Department, British Council) writes: We think that your readers may like to know that as a result of the overwhelming response to the appeal for used copies of medical journals (which was published in the *British Medical Journal*, November 24, p. 1291, after a suggestion made by Sir Clement Price Thomas following his visit to Yugoslavia) the total number of medical periodicals already forwarded to Belgrade is about 4,600. Our representative, in acknowledging them, writes that Dr. Djukanovic, the Yugoslav Deputy Minister of Health, upon being informed of this valuable gift, immediately sent two members of the committee dealing with the distribution of medical periodicals in Yugoslavia to examine the consignment. The two doctors were clearly delighted by the sight of so many much-needed British journals and at once agreed to arrange for their distribution throughout the country, promising that priority should be given to professors of university medical and surgical clinics, as suggested by Sir Clement Price Thomas, and that hospitals and surgeons whom Sir Clement visited during his recent operating tour should also be included in the distribution. Because of the large number of journals received, it has been impossible to acknowledge them all individually, and we should be most grateful if you would convey our thanks to the many donors through the columns of the *British Medical Journal*.

### Frequency

Dr. R. A. P. CORKERY (West Drayton) writes: . . . I was brought up in perhaps a pedantic school of medicine and was always taught that "frequency of micturition" is normal and healthy, but that "increased frequency" is not. Surely, Sir, anything that occurs periodically can be termed "frequent," and can still be within normal limits. We can talk of frequency of meals, frequency of surgeries, frequency of holidays—they become abnormal only when this normal frequency is altered. If the term "frequency" is construed as I think it should be . . . the only cause of frequency of micturition is the presence of a sufficiency of fluid in the bladder.

### Staining of Brain Tumours

Dr. I. EBAN (London, W.1) writes: In your annotation on "Locating Brain Tumours with Isotopes" (February 23, p. 428), 1948 is the earliest date which you give in connexion with the staining of brain tumours by intravenous injection of a suitable dye. For the sake of historical accuracy I would like to point out that as far back as 1931 brain staining was already being done by Friedemann and Elkeles. This fact is mentioned with some detail in the *Proceedings of the Royal Society of Medicine* for January, 1943 (pp. 137–140).

### Correction

In the account of the life of the late King, which was published in the *Journal* of February 16 (p. 386), the name of Sir Morton Smart, G.C.V.O., D.S.O., M.D., was omitted, through an error which we much regret, from the signatories of the bulletin (issued on November 23, 1948) announcing that the King was suffering from obstruction to the arterial circulation of the legs.

# Obituary

## A. R. PARSONS, M.D., F.R.C.P.I.

Alfred Robert Parsons, who died at his home in Dublin on January 24, was born in 1864, and after a brilliant career at Trinity College, Dublin, graduated M.B., B.Ch. in 1888. He won the medical scholarship and medical travelling prize, and continued his studies in Vienna and Berlin. In 1894 he was appointed visiting physician to the Royal City of Dublin Hospital, a position he held till his death, and rapidly became one of the leading physicians in Dublin and an outstanding clinical teacher. In 1894 also he was elected a Fellow of the Royal College of Physicians of Ireland, of which he was senior Fellow for many years.

Throughout the years his mind remained as keen and as active as ever, and his bodily vigour showed little sign of impairment. He never retired, but continued his full hospital work to the end, taking his usual clinical class the week before he died. For 58 years he was a fascinating clinical teacher, one who could hold the attention of students and stimulate their interest. He taught them to observe accurately, to examine a patient carefully and critically, to evaluate what was found, and to be self-reliant, but not cocksure, in judgment. A diagnosis was held in almost tantalizing suspense until all the facts on which it was based had been thoroughly worked out. Parsons saw many changes in medicine, but his reliance on symptoms and, above all, on physical signs as the sure foundation for diagnosis did not change. Although pathology, bacteriology, serology, radiology, biochemistry, and all the other present-day aids to diagnosis were used by him fully, he was not carried away by any uncritical enthusiasm for what was new. In treatment he showed no hesitation in abandoning the elegant potions and pills of the 'nineties in favour of the synthetic chemicals and antibiotics of to-day, and was always eager to use a new remedy, though he tried out each one carefully and experimentally before passing judgment upon it.

During the first world war Parsons served in the R.A.M.C. He was on the staff of 83 General Hospital in France with the rank of lieutenant-colonel.

Parsons took a keen interest in scientific medical meetings. He was an original member of the Association of Physicians of Great Britain and Ireland, and was a past president of the Royal Academy of Medicine in Ireland, and a member of the "Bi" Club. It was a great pleasure to hear him read a paper or to see him demonstrate a rare clinical case. The thorough investigations, the scientific clarity, and the dramatic presentation were all admirable.

Dublin has lost its oldest and most beloved clinical teacher. He was a steadfast and loyal colleague, a skilful and warm-hearted physician, a courteous and modest man. His sense of duty, his tenacity of purpose, and his rigid moral values remained unchanged throughout his life. Thousands of his former students in all parts of the world will always remember "Alfie" Parsons with gratitude, affection, and veneration.—V. M. S.

## Sir DOUGLAS SHIELDS, M.D., F.R.A.C.S.

Sir Douglas Shields, who began his career as a surgeon in Australia and later built up a big practice in London, died at Merstham, Surrey, on February 22, aged 73.

Douglas Andrew Shields was born in Melbourne on July 21, 1878. His father was a much-respected Scottish doctor holding an administrative position in that city. Douglas Shields studied medicine at Melbourne University, to which he won a scholarship and where he was first among those who obtained first-class final honours. He graduated M.B., Ch.B. in 1897 and proceeded M.D. in 1901. For a time he practised in a country district, but soon returned to Melbourne on his appointment to the staff of St. Vincent's Hospital, where he became a senior surgeon. Shortly after his return from a trip to London he was appointed consulting surgeon to the Governor-General of Australia (at that time the Earl of Dudley).

Two or three years before the first world war Shields decided to leave Australia and settle in London, where he established a private hospital in Park Lane. During the war this hospital was given up to the treatment of wounded officers. For a time he worked as a surgeon at a hospital in Wimereux in France. He was knighted for his wartime services in 1919, and three years later given the honorary degree of LL.D. at St. Andrew's University during the Rectorship of J. M. Barrie. After the war Shields's surgical practice continued to expand, and for a time he worked in association with Sir Alfred Fripp. His attractive personality and good appearance made him a much-sought-after man. His professional reputation was well deserved, for he was a neat and careful operator and kind and attentive to his patients.

Sir Douglas Shields married Mary Ellen, daughter of the late Charles Shirrefs, in 1900. His wife died in 1939, and he is survived by a son, who is also a medical man.

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Dr. ARCHIBALD MACEWAN died on January 31, after a brief illness, aged 51. A native of Lochgilphead, Dr. MacEwan was born on October 2, 1900, and was educated at the University of Glasgow, where he graduated M.B., Ch.B. in 1925. In the same year he joined his cousin, the late Dr. John A. Thomson, in general practice at Hamilton, where he practised until his death. He was on the Crown list of medical examiners, and on many occasions gave evidence in the High Court and Sheriff courts, where he was regarded by Bench and Bar as a scrupulously fair expert. He was appointed a medical referee for the Sheriffdom of Lanark in 1945, and, until the abolition of the Poor Law system in 1948, when the National Assistance Act was placed on the Statute Book, was a certifying officer under the Lunacy Acts. During the war he was keenly interested in civil defence, and gave much of his leisure time to the training of first-aid and ambulance units. A quiet, kindly man who inspired confidence in his patients, he will be sadly missed by them and by his many friends. He is survived by his wife and son.—A. N. D.

The death on February 16 of Dr. J. W. LOGIE removed a general practitioner of the old school and one of distinguished ability. Born in 1869 of Orcadian stock, John William Logie was educated at Glasgow High School and at the University of Glasgow, where he had a notable career as a student. He graduated M.B., C.M., with high commendation, in 1891, and, after serving as a resident house-surgeon at the Victoria Infirmary and as resident assistant physician at the Belvidere Fever Hospital, Glasgow, he was for some years in practice in Partick. A period at sea, as a ship surgeon, followed for health reasons. He resumed general practice first at Pontyclun, Glamorganshire, and then at Rothes, in Morayshire. During the war of 1914–18 he was in charge of the Red Cross Hospital at Pontyclun, receiving Queen Alexandra's Medal for his services. Failing health in the early 1930s led to his retirement to Aberdeen, where during the last war he served regularly on medical boards. Throughout his retirement Dr. Logie retained an alertness of mind and crispness of phrase, both of which gave great pleasure to his many friends, professional and lay. Proud of his profession, though critical of some recent changes, he maintained contact to the last with current developments in medicine. His insight and robust good sense, informed by wide experience as a practitioner, were greatly valued by younger colleagues. In August last Dr. and Mrs. Logie celebrated their golden wedding. He is survived by his widow, a son, Mr. Norman J. Logie, the Aberdeen surgeon, and a daughter, who is the wife of Dr. George I. Davidson, of Aberdeen.—R. M. N.

Dr. ALFRED HOWARD PARKINSON died at Wolverhampton on February 16, aged 76. After a childhood spent partly in France he began his working life as a teacher of science in Knaresborough, Yorkshire. He took the London B.Sc., with honours, in 1901, and then his interest turned to medicine, and he graduated M.B., B.S. in 1906, taking the Cambridge D.P.H. in 1909 and proceeding M.D. in 1911. After periods in Leeds and in the east end of London he was appointed by the newly constituted Staffordshire, Wolverhampton, and Dudley Joint Board for Tuberculosis to be tuberculosis officer to the Stafford area, which included Groundslow Sanatorium, at Tittensor, Stoke-on-Trent. Except during his service in the R.A.M.C. in the war of 1914–18, he continued to act in this capacity until March, 1947. His work was his whole life, and after his retirement he kept himself fully employed by acting as locumtenent for many colleagues in Staffordshire. He was an indefatigable worker and could not be persuaded to take life easily. His mind was so alert and his interest so keen that during the past few years he frequently expressed regret that he could not begin all over again now that so many advances had been made in the treatment of pulmonary tuberculosis. Dr. Parkinson was a past president of the Midland Tuberculosis Society, and in his presidential address a few years ago he reviewed the many changes in treatment which had occurred in his lifetime. During these long years of service his wide clinical knowledge was willingly given for the assistance of patients and colleagues, and it was rendered the more valuable by his unfailing courtesy and kindness. His generous human sympathy was particularly helpful in the type of work he had chosen. He was held in affectionate esteem by his colleagues in Staffordshire, with many of whom his association had been very close. Many patients will mourn his passing, for he was indeed a good physician. He had continued at work until three weeks before his death, and gave in only when illness compelled him to do so. He is survived by one married daughter.—G. R.

Lieutenant-Colonel F. M. GRAHAM, late I.M.S., who died in Manchester on February 17, after a short illness, was born in Bangalore, Southern India, on March 8, 1892, the eldest son of William S. and Florence E. Graham. Frederick Montague Graham received his medical education at Madras Medical College, and took his diploma in 1914. He saw active service in the Middle East during the war of 1914–18, being twice mentioned in dispatches, and he served also in Afghanistan in 1919 and in the Calicut riots of 1921. In 1922 he continued his medical studies at King's College Hospital, London, taking the M.R.C.S., L.R.C.P. in 1924. He returned to India in that year and joined the civil medical service of Madras, where he was lecturer in surgery and medical jurisprudence at Stanley Medical College from 1924 to 1929. Later he held hospital appointments in Madras Presidency, and he joined the staff of Government House, Madras, in 1934. In 1944 he was recalled to active service in the Middle East Paiforce (Persia, Afghanistan, Iraq), in which he served throughout the last war. He retired from the I.M.S. in 1947.

A colleague writes: In January, 1949, Graham was appointed a medical officer of the Ministry of Pensions, and, though he was to hold the post only some three years, his quiet ways and endearing qualities made a particular impression on his colleagues, and his loss is most keenly felt. He was quite selfless and had a strong and abiding religious faith. Lieutenant-Colonel Graham is survived by his widow, a son, and a daughter. His son is demonstrator in pathology at King's College Hospital and his daughter is training as a radiologist at the Manchester Royal Infirmary. The sympathy of his many friends, professional and lay, is extended to them in their grievous loss.

Cumberland has lost a wise and skilful surgeon by the death, on February 21, of Mr. GORDON NEILL at the early age of 34. Andrew Gordon Currie Neill did well at Sedbergh School and at the University of Edinburgh, where he graduated M.B., Ch.B., with honours, in 1941. After serving as house-surgeon and house-physician at Cumberland Infirmary, Carlisle, he became resident surgical officer at Hull Royal Infirmary. He was elected F.R.C.S.Ed. in 1943 and returned to Cumberland Infirmary as resident surgical officer, becoming honorary assistant surgeon in 1947. Early in his professional career Neill came under the influence and guidance of Mr. J. N. J. Hartley, and was at an unusually early age a surgeon of wide experience, mature judgment, and quite exceptional technical achievement. A modest manner lightly cloaked an inner strength which gave his patients confidence, so that they became his friends. He accomplished a vast amount of work, but never seemed to be hurried. He had a serene temperament, a prodigious memory, and a genuine interest in the welfare of his patients, for each of whom, no matter how weary he may have been, he had a kind word. He abhorred the idea of destructive surgery and turned his attention more and more to plastic surgery, at which he soon became adept. Neill was well read and had an interest in many subjects, not least in history and the classics. To an extraordinary degree he had developed the faculty of reaching the core of a problem through a mass of detail. This enabled him to reach a reasoned decision in his work and helped to make him such an admirable secretary to the medical board and later to the medical advisory committee. He was a man of firm beliefs and opinions, but he saw the other man's point of view clearly enough never to irritate nor to give offence in a discussion. The main memory he leaves is one of quiet equanimity and unobtrusive hard work by which he achieved his aim—to become an "operating physician." In spite of much mental and physical suffering during the last few weeks of his life Neill remained calm, cheerful, and appreciative of the little that could be done to help him. To his widow and two baby daughters and to his parents his many friends extend their deep sympathy. —T. McL. G.

Dr. RAYMOND HENRY SHAW, who died in South Kensington on February 24 after a long illness, was born in Newcastle-upon-Tyne in November, 1866. His father, Benjamin Shaw, was a schoolmaster there, and his son was educated under him at Singleton House School. He studied medicine at Durham University, where he graduated M.B. with honours and B.S. in 1889: he had four scholarships there between 1886 and 1889, and he proceeded M.S., with first-class honours, in 1892, after holding an appointment as house-surgeon at the Royal Infirmary, Newcastle-upon-Tyne. About 1894 he joined the late Dr. Ryley in practice at Great Yarmouth, where he remained until he retired in 1932. He volunteered for the R.A.M.C. in the 1914–18 war, but was not accepted; and indeed his work at Yarmouth Hospital was probably of more advantage to his country than active service might have been. His house was hit during that war by a bomb, and his young daughter was wounded. Shaw had decided leanings towards surgery, though he was first and foremost a highly competent and successful general practitioner. He was surgeon to the Yarmouth General Hospital for about 24 years—much of that time senior surgeon. He was also surgeon to the Yarmouth police and medical officer to the Guardians' Infirmary and Children's Homes. He objected strongly to Lloyd George's National Health Insurance Act and led the opposition to it in his district. He was disappointed that it was finally brought into force and resigned his membership, never afterwards rejoining the Association. He married Miss Katharine Robson, of Darlington, in 1904. His wife and daughter survive him; his son, Raymond Thorndike Shaw, died of wounds while serving as a lieutenant-commander in the Royal Navy in 1942. After retiring Shaw made a good many voyages to the Far East as a ship surgeon, and to his great credit turned out again for this work during the last war. In 1942 his ship was hit by bombs off the coast of Scotland; he stayed in the ship until she was beached, incurring frostbitten toes as a result—a fine record for a man of 75.

# Medico-Legal

## PRIVATE PATIENT'S FEES

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

In an action tried in the Willesden County Court[1] Judge Sir Henry Braund reduced the amount of a doctor's claim for fees from a private patient in respect of infra-red treatment and injections from £39 7s. 6d. to £25 15s. The claim was for 24 visits at 1½ guineas a time, with an initial visit at 3 guineas including a blood test.

The doctor said in evidence that he did not discuss the amount of his fee with the patient other than to say that his charge would be moderate as he knew she was not well off. He belonged to an old-fashioned generation of doctors who did not like to speak of money.

This case illustrates the principles of the English law of contract which affect the remuneration of doctors in private practice. The principles are of general application in the profession with the exception that a condition of admission to the Fellowship of the Royal College of Physicians is that a Fellow of the College may not sue for his fees.

In law the doctor in private practice can expressly agree with his patient any fee that is satisfactory to them both. If the patient will not pay, the doctor is entitled to sue him upon the express agreement for the amount agreed, and will recover that amount. Where the amount is expressly agreed it does not matter whether the court thinks it is too large or too small. It is for the parties to make their bargain, and the court will enforce the bargain they have made.

Alternatively, the patient may ask the doctor to treat him and the doctor may do so without either of them saying a word about the cost of the treatment. In that event the law implies a promise by the patient to pay a reasonable amount for the treatment, and, if the patient does not pay, the doctor is entitled to sue him upon this implied promise. The cause of action is called in the law of contract a *quantum meruit*. It is then for the court to say how much is "reasonable." The court in doing so applies objective considerations, and what the doctor or the patient may think is a reasonable fee is only an element in the evidence—to be taken into consideration together with any other evidence which may be adduced. The court will treat the evidence of expert witnesses called to say what in their opinion is a reasonable charge for the treatment given purely as a matter of opinion, giving it such weight as the expert who gives it can command. But it is for the court, after all the evidence has been given, to decide what is a reasonable amount. As Judge Sir Henry Braund observed in the Willesden case, what is reasonable in Harley Street may not be reasonable in Finchley Road.

If any doctor finds himself under the unpleasant necessity of having to sue for his fees, the advantages of having an express agreement to sue upon are therefore very great.

[1] *Hampstead and Highgate Express*, February 23, 1951.

# Medical Notes in Parliament

### The National Drug

During the debate on the second reading of the Customs and Excise Bill on February 19 Dr. BARNET STROSS referred to the clause (229) dealing with tea, coffee, cocoa, etc. This contained paragraphs referring to the analysis of imported tea, which may have other substances added to it. He said that for 200 years it had been a national belief that to drink tea was good for one. He worked out that on an average consumption of 3 oz. (85 g.) of tea a week the total national intake of pure caffeine was about 50 million pounds a year (22.7 million kg.). There were about one and a half grains (100 mg.) of pure caffeine in the average strong cup of tea. Some people thought nothing of drinking 10 cups of tea a day, which would give them 15 gr. (1 g.) of caffeine. Yet the maximum medicinal dose of caffeine a day was 5 gr. (315 mg.). People who took an excessive amount of caffeine through their tea were the truest type of drug addicts, because caffeine was a true cerebral stimulant. The amount of alcohol found in beer and cider was not stimulating, and was ultimately depressing.

Mr. JOHN BOYD-CARPENTER, replying to the discussion, made no comment on this point.

### Pneumoconiosis and Byssinosis

Mr. R. H. TURTON on February 20 moved that the House approve the Draft Pneumoconiosis and Byssinosis Benefit Scheme, 1952. He said that the Act of 1951 had set out provisions which this scheme embodied. It put uncompensated men and their dependants in the same position as if they were entitled to workmen's compensation for total disablement, or death from either disease. Whether a man was totally disabled or not, and whether death was a direct result of the disease, would have to be decided by a medical board.

Dr. EDITH SUMMERSKILL remarked that the scheme dealt only with the totally disabled, and hoped there would be further investigation in regard to the partially disabled. The scheme could be used for other diseases, and she asked Mr. Turton to consider such diseases as epithelioma.

Miss MARGARET HERBISON said that she and other members representing mining constituencies constantly had miners telling them that their own doctor diagnosed pneumoconiosis. Such cases were sent to Edinburgh Royal Infirmary, where a specialist, after an *x*-ray examination, decided that a man had pneumoconiosis, and he was thereafter sent to the medical board, which, in a number of cases, decided the man was not suffering from the disease. It was impossible to make a miner believe that he had a square deal when a specialist gave one decision and the medical board a different one. Mr. EDWARD DAVIES agreed that there was dissatisfaction because men felt that, while they had courtesy and an excellent service from the medical boards, there was often a conflict among medical men themselves on the diagnosis. The men must be assured that whoever had the responsibility of making a final decision in these matters was of the highest medical reputation.

Dr. BARNET STROSS said cases were known in which, because the disease was progressive, refusal in one year was followed by acceptance in the next, and asked whether that procedure would be continued. Mr. TURTON said that it would, and after further discussion he replied to the debate, reaffirming that on the interpretation of total disability and the question whether death arose from pneumoconiosis the decisions of medical boards would be final in future as at present. The House then agreed with the motion that the scheme be approved.

### Cost of Patients' Food

Mr. T. F. PEART on February 25 asked the Minister of Health to give instructions to regional hospital boards that no economies should be made in hospital expenditure on patients' food. Mr. H. F. C. CROOKSHANK answered that he could not do as suggested because this was within the discretion of hospital authorities, who might find it possible to secure some measure of economy without prejudicially affecting patients' diets.

### Health Service Appliances

Mr. H. F. C. CROOKSHANK told Mr. A. BLENKINSOP on February 25 that the great variety of appliances supplied to patients through the hospitals in England and Wales during the 12 months ending December 21, 1951, could be broadly grouped under the following headings, for which the numbers and cost were:

| Appliance | No. Supplied in 12 Months | Cost £ |
|---|---|---|
| New surgical footwear | 46,465 | 395,000 |
| Appliances for lower limbs | 28,280 | 156,000 |
| Trusses | 19,085 | 35,000 |
| Abdominal appliances | 107,282 | 375,000 |
| Suprapubic and kindred appliances | 15,570 | 55,000 |
| Appliances for upper limbs | 3,593 | 12,500 |
| Spinal supports | 58,656 | 410,000 |
| Head and neck appliances | 11,732 | 58,000 |
| Elastic hosieries | 35,520 | 80,000 |
| Wigs | 9,756 | 125,000 |
| Prostheses (artificial breasts, ears, eyes, noses, etc.) | 11,715 | 29,000 |
| Artificial arms | 1,937 | 51,500 |
| ,, legs | 11,383 | 330,000 |
| ,, arm appliances | 4,058 | 15,000 |
| ,, eyes | 7,682 | 12,000 |
| Invalid chairs and tricycles | 10,108 | 685,073 |

### Safety on the Roads

Brigadier FRANK MEDLICOTT on February 28 asked the Home Secretary to take steps to make a blood test for alcohol compulsory in the case of the driver of a vehicle involved in an accident which caused death or injury to any person. Sir DAVID MAXWELL FYFE replied that the proposal involved a serious encroachment on personal liberty. On his present information he would not feel justified in proposing such a fundamental alteration in the law.

Mr. J. S. MACLAY stated that, in the absence of evidence that defective eyesight of drivers was an important factor in road accidents, he did not think that further steps were necessary to ensure that the eyesight of drivers was sufficiently good to prevent accidents. He added that a requirement that some individuals should wear glasses while driving would be unenforceable.

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*Artificial Limbs.*—No charge will be made for artificial limbs, for most surgical aids needed by the seriously disabled, nor for any appliances supplied to patients who are resident in hospital rehabilitation centres.

*"Roter" Tablets.*—Mr. H. F. C. Crookshank has stated that these proprietary tablets for treatment of peptic ulcer could not be regarded as essential. He was therefore unable on medical grounds to recommend that import restrictions should be waived.

# Vital Statistics

### Smallpox in Lancashire

The total number of cases of variola minor ascertained up to the morning of Tuesday, March 4, was 70 (64 in Rochdale, 5 in Milnrow, and 1 in Heywood). All these cases are associated with the original focus in Rochdale. Thirty-seven cases are in hospital. The remainder are no longer infectious. Until March 3 the cases had occurred in the direct line of contact. On March 3, 4 cases were found which cannot at present be associated with previous cases. There is no report of smallpox in any other part of the country.

### Industrial Accidents and Diseases

The number of workpeople (other than seamen) in the United Kingdom whose deaths from accidents in the course of their employment were reported in January was 131, compared with 99 in the previous month and 140 (revised figure) for January, 1951. Of these deaths 41 were underground in coal mines and 18 were in building operations.

The following cases of industrial disease were notified during the month: lead poisoning 4, aniline poisoning 4, toxic anaemia 1, anthrax 2, epitheliomatous ulceration 11, chrome ulceration 20 (chromium plating 10); total 42. There were no deaths (*Ministry of Labour Gazette*, February, 1952).

During 1951, 485 persons were killed in coal mines and 1,942 seriously injured, as compared with 493 and 2,020 (revised figure) respectively in 1950.

### Infectious Diseases

A rise in the number of notifications was recorded in England and Wales for the week ending February 16 for measles 555, scarlet fever 65, and dysentery 44, while the only fall of any size was 136 for whooping-cough.

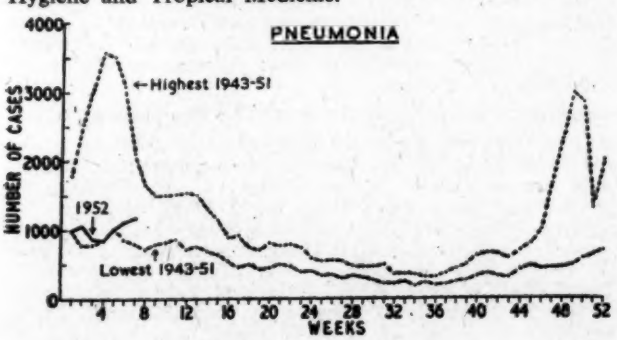
A small rise in the incidence of measles was recorded in many areas. The largest rises were Cheshire 166 and Middlesex 98, while the only large exception to a rising trend was a fall of 172 in Lancashire. With the exception of a small rise in London and the adjacent counties, the incidence of scarlet fever remained practically constant. The only large variation in the local trends of whooping-cough was a decline of 87 in Yorkshire West Riding. The notifications of diphtheria were 14 more than in the preceding week. The rise in diphtheria was chiefly contributed by Lancashire with an increase of 8; 10 cases were notified in Darwen M.B., where 5 cases were reported in the preceding week.

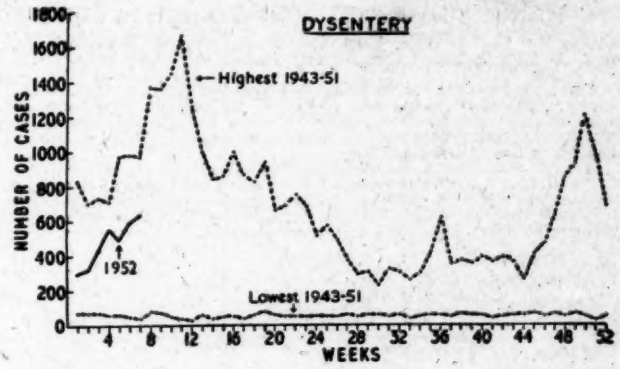
The number of notifications of acute poliomyelitis were 2 more for paralytic and 1 more for non-paralytic cases than in the previous week. The only administrative area with more than 1 case was Sussex, Southwick U.D., with 2 cases.

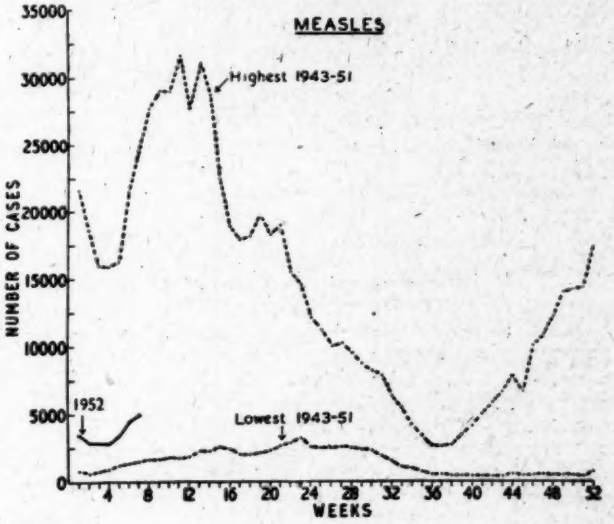
An important factor in the increased incidence of dysentery was a rise of 86 in the number of notifications in Lancashire. A further 62 cases were notified in the outbreak in Norwich C.B., bringing the total notifications for this centre up to 984. The largest returns during the week were Lancashire 157 (Oldham C.B. 46, Manchester C.B. 36, Chadderton U.D. 11, Bolton C.B. 10); London 83 (Hackney 23, Wandsworth 11); Norfolk 72 (Norwich C.B. 62); Middlesex 51 (Willesden M.B. 18); Yorkshire West Riding 34 (Leeds C.B. 13); and Surrey 33.

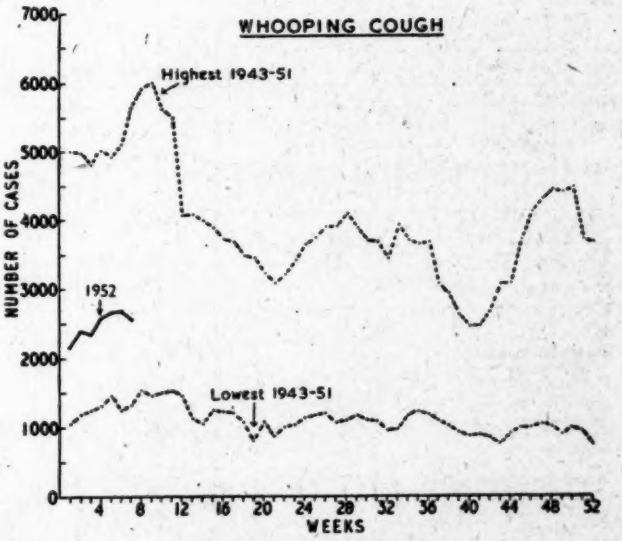
### Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported during the nine years 1943–51 are shown thus --------, the figures for 1952 thus ————. Except for the curves showing notifications in 1952, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.




### Week Ending February 23

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,613, whooping-cough 2,648, diphtheria 37, measles 5,734, acute pneumonia 1,143, acute poliomyelitis 14, dysentery 591, paratyphoid fever 6, and typhoid fever 3.

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**Census Changes.**—Population figures obtained at the 1951 census show that there are now 160 great towns (population over 50,000) in England and Wales instead of 126, and 17 principal towns (population over 30,000) in Scotland instead of 16. Therefore the 1952 figures for deaths, births, and stillbirths in these countries, published weekly in our Table, cannot be accurately compared with those for 1951, which are printed in italics. The 160 great towns in England and Wales have a population about 10% higher than the 126.

## INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending **February 16** (No. 7) and corresponding week 1951.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

| CASES in Countries and London | 1952 Eng. & Wales | 1952 Lond. | 1952 Scot. | 1952 N. Ire. | 1952 Eire | 1951 Eng. & Wales | 1951 Lond. | 1951 Scot. | 1951 N. Ire. | 1951 Eire |
|---|---|---|---|---|---|---|---|---|---|---|
| Diphtheria | 48 | 4 | 15 | 0 | 1 | 40 | 8 | 15 | 0 | 1 |
| Dysentery | 636 | 83 | 62 | 5 | 1 | 972 | 42 | 266 | 6 | 1 |
| Encephalitis, acute | 3 | 0 | 1 | 0 | | 10 | 1 | 0 | 0 | |
| Enteric fever: | | | | | | | | | | |
| Typhoid | 3 | 0 | 0 | 1 | 0 | 4 | 2 | 0 | 0 | 5 |
| Paratyphoid | 3 | 0 | 0 | 0 | 1 (B) | 11 | 1 | 1 (B) | 0 | |
| Food-poisoning | 114 | 11 | | 1 | | 99 | 29 | | 1 | |
| Infective enteritis or diarrhoea under 2 years | | | | 10 | 17 | | | | 13 | 10 |
| Measles* | 4,980 | 103 | 365 | 185 | 107 | 24,585 | 3178 | 219 | 52 | 67 |
| Meningococcal infection | 51 | 5 | 18 | 4 | 5 | 50 | 11 | 17 | 3 | 1 |
| Ophthalmia neonatorum | 27 | 5 | 8 | 0 | | 24 | 3 | 7 | 0 | |
| Pneumonia† | 1,175 | 40 | 406 | 15 | | 2,115 | 107 | 305 | 25 | 93 |
| Poliomyelitis, acute: | | | | | | | | | | |
| Paralytic | 13 | 1 | 4 | 3 | 1 | 18 | 0 | 3 | 0 | 0 |
| Non-paralytic | 7 | 0 | | | | 6 | 1 | | | |
| Puerperal fever‡ | 226 | 32 | 16 | 1 | | 67 | 9 | 11 | 4 | 1 |
| Scarlet fever | 1,481 | 142 | 219 | 15 | 44 | 904 | 68 | 159 | 25 | 52 |
| Tuberculosis: | | | | | | | | | | |
| Respiratory | | | 136 | 35 | | | | 178 | 37 | |
| Non-respiratory | | | 27 | 3 | | | | 22 | 3 | |
| Whooping-cough | 2,550 | 101 | 289 | 125 | 98 | 5,662 | 404 | 778 | 52 | 97 |

| DEATHS‡ in Great Towns | 1952 Eng. & Wales | 1952 Lond. | 1952 Scot. | 1952 N. Ire. | 1952 Eire | 1951 Eng. & Wales | 1951 Lond. | 1951 Scot. | 1951 N. Ire. | 1951 Eire |
|---|---|---|---|---|---|---|---|---|---|---|
| Diphtheria | 0 | 0 | | 0 | 0 | *0* | 0 | *0* | 0 | 0 |
| Dysentery | 0 | 0 | | 0 | | | 0 | | 0 | |
| Encephalitis, acute | | 0 | | | 0 | | 0 | | | 0 |
| Enteric fever | 0 | 0 | | 0 | | *0* | 0 | *0* | 0 | |
| Infective enteritis or diarrhoea under 2 years | 24 | | 1 | 1 | 0 | *21* | | *3* | 1 | 4 |
| Influenza | 40 | 6 | 4 | 1 | 0 | *492* | 52 | *14* | 11 | 50 |
| Measles | | 0 | | 0 | 0 | | 2 | *0* | 0 | 0 |
| Meningococcal infection | | 0 | | | | | 1 | *1* | | |
| Pneumonia | 338 | 48 | | 14 | 9 | *535* | 68 | | 17 | 18 |
| Poliomyelitis, acute | 3 | 0 | | | 0 | *4* | 0 | | | 0 |
| Scarlet fever | | 0 | | 0 | 0 | | 0 | *0* | 0 | 0 |
| Tuberculosis: | | | | | | | | | | |
| Respiratory | 154 | 25 | 39 | 7 | 9 | *183* | 27 | *22* | 6 | 17 |
| Non-respiratory | | 1 | 5 | 0 | 0 | | 3 | *6* | 1 | 6 |
| Whooping-cough | 3 | 0 | | 1 | 0 | *14* | 3 | *2* | 0 | 1 |
| Deaths 0–1 year | 300 | 24 | 37 | 11 | 17 | *301* | 39 | *33* | 6 | 18 |
| Deaths (excluding stillbirths) | 6,790 | 1031 | 809 | 147 | 203 | *7,688* | 1162 | *765* | 158 | 315 |
| LIVE BIRTHS | 7,440 | 1100 | 856 | 211 | 356 | *7,104* | 1208 | *905* | 242 | 361 |
| STILLBIRTHS | 202 | 20 | 28 | | | *179* | 18 | *30* | | |

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
‡ Includes puerperal pyrexia.
‡ See "Census Changes" on previous page.

# Universities and Colleges

## UNIVERSITY OF CAMBRIDGE

The Senate on February 23 approved an Address to Her Majesty the Queen expressing condolence on the death of His Majesty King George VI and the respectful homage of the University to Her Majesty on her succession to the Throne. An Address of condolence to Her Majesty Queen Elizabeth the Queen Mother was also approved.

The following medical degrees were conferred on February 23:

M.CHIR.—*G. M. Lunn.

M.B., B.CHIR.—*R. F. Pearson, *Katharine C. Paterson, I. H. Kerr, C. McC. Campbell.

The following candidates have been approved at the examination indicated:

M.CHIR.—T. Fenwick, T. W. Rowntree, R. N. Ticehurst, W. Waugh.

*By proxy.

## UNIVERSITY OF LONDON

The following have been recognized as teachers of the University in the subjects indicated in parentheses: *St. Bartholomew's Hospital Medical College*, Dr. H. F. Brewer and Dr. H. Lehman (Pathology); Dr. C. S. Nicol (Venereal Diseases); Mr. A. S. Philps (Ophthalmology); Dr. B. F. Russell (Dermatology). *Postgraduate Medical School of London*, Dr. E. G. L. Bywaters and Dr. J. F. Goodwin (Medicine); Mr. M. R. Ewing, Mr. R. H. Franklin, Mr. G. C. Knight, and Mr. R. Shackman (Surgery). *National Institute for Medical Research*, Dr. O. G. Endholm (Physiology). *Charing Cross Hospital Medical School*, Dr. J. A. Gorsky (Forensic Medicine); Dr. S. Shaw (Pathology). *University College Hospital Medical School*, Dr. W. Grossman (Dental Surgery); Miss Doreen Nightingale (Surgery). *St. George's Hospital Medical School*, Mr. J. J. Richmond (Radiology). *St. Mary's Hospital Medical School*, Mr. I. G. Robin (Oto-rhino-laryngology); Dr. K. W. Cross (Physiology). *University College*, Dr. R. D. Harkness (Physiology); Dr. S. E. Dicker (Pharmacology). *School of Pharmacy*, Dr. Eleanor J. Zaimis (Pharmacology). *St. Thomas's Hospital Medical School*, Dr. J. R. Napier (Anatomy).

Dr. B. A. D. Stocker has been granted probationary recognition as a Teacher of Pathology at the London School of Hygiene and Tropical Medicine for two years from December, 1951.

The following, who are already recognized teachers at undergraduate medical schools, have been recognized also as teachers at the postgraduate institutes and in the subjects indicated: *Institute of Obstetrics and Gynaecology*, Mr. I. M. Jackson (Obstetrics and Gynaecology). *Postgraduate Medical School of London*, Mr. C. H. Gray (Surgery).

The William Julius Mickle Fellowship for 1951–2 has been awarded to Rosalind Venetia Pitt Rivers, M.Sc., Ph.D., in respect of the investigations into biochemical and physiological problems connected with thyroxine and kindred substances which she has carried out during the past five years.

In Convocation on January 19 it was announced that Drs. C. A. H. Franklyn and W. J. O'Donovan had been re-elected as representatives of the Faculty of Medicine on the Standing Committee of Convocation for a period of three years.

## UNIVERSITY OF SHEFFIELD

At a meeting of the University Council held on February 18 it was decided to send a Loyal Address to Her Majesty Queen Elizabeth II.

The Council has appointed S. R. Elsden, Ph.D., Senior Lecturer in Microbiology, to be in charge of a separate Department of Microbiology, the arrangements to take effect from October 1.

The Council received the resignations of Dr. L. G. Cook, Honorary Lecturer in Bacteriology, on his return to New Zealand, and Dr. B. A. E. Johns, Lecturer in Anatomy, who is returning to clinical work, and thanked them for their services to the University.

## ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH

At a quarterly meeting of the college, held on February 5, with the President, Dr. W. A. Alexander, in the chair, the following were elected Fellows of the College: C. M. Fleming, W. Henderson, H. E. Seiler, I. H. L. Gillies, W. H. Galloway.

The following were elected Members of the College: W. Boyd, Irene P. Rowlands, S. Stein, R. Orton, J. N. Armour, G. W. Reid, V. D. Bayliss, S. Prakash, H. McD. Forde, N. Gordon, R. Prasad, B. M. Kher, L. Albert, J. Gant, M. P. Mehrotra, Jean C. Taylor, H. Black, A. C. Douglas, P. C. MacGillivray, M. A. Botawala, S. D. Store, R. S. M. D. Inch.

# Medical News

**Anglo-French Medical Exchange Bursaries.**—The French Embassy in London has recently negotiated an agreement between the Institut National d'Hygiène, Paris, and the Ciba Foundation, London, for an exchange of short-term grants to enable young British and French doctors to visit each others' countries and study special techniques. The grants, which will have a minimum value of £50 a month tax-free, will normally be awarded for a minimum of two and a maximum of six months. The bursaries are intended primarily for doctors wishing to obtain clinical experience, but they can also be awarded for medical research. Candidates must have had at least one year's experience after qualification and must indicate in their application the place in which they wish to carry out their study. Candidates for the French bursaries must be British subjects and citizens of the United Kingdom. They will be chosen by a selection board. Applications for the 1952 bursaries should be made before March 31 to Dr. G. Wolstenholme, Ciba Foundation, 41, Portland Place, London, W.1, from whom application forms can be obtained.

**Manchester Medical Society.**—On January 28 the inaugural meeting of the new section of general practice was held with Dr. R. R. Kerr in the chair. This section has been formed at the request of a large number of Manchester general practitioners with the object of holding meetings at times convenient to them and to enable them to have lectures and discussions on problems closely connected with general practice. Any general practitioner in Manchester and district who is interested in becoming a member is invited to communicate with the honorary secretary of the Manchester Medical Society at Manchester University.

**Liverpool Medical Institution.**—At a meeting on February 14 Dr. R. B. MacKenna read a paper entitled "Some Problems in Dermatology," and discussed four problems in dermatology. Infantile eczema, particularly the atopic type of that malady, was a matter in which an ecological survey would be likely to be helpful. Psoriasis was probably a dermadrome—that is, the eruption on the skin was but the cutaneous part of a syndrome in which the endocrine system was principally involved. Thirdly, cross-sensitization phenomena were considered, and finally he stated that work done by Dr. Ida Macalpine in his department at St. Bartholomew's Hospital indicated that certain cases of recurrent dermatitis of the eyelids, which hitherto had been widely regarded as being invariably due to allergic sensitivities, were sometimes manifestations of nervous stress and were not really manifestations of allergy.

In the discussion which followed Dr. J. M. H. Smellie said he would like to mention a case of arthritic psoriasis, which Dr. MacKenna had seen in consultation with his (Dr. Smellie's) old colleague, Dr. Gerald Wallace. Dr. Smellie was asked to help in mobilizing her shoulders, elbows, hips, and knees to make her fit on a frame, prior to her admission to hospital. There the first injection of "mercaloid" produced albuminuria and vomiting, and the second made her so much worse that she was taken home. Her father, resigned to her being a permanent invalid, had doors fitted to the back of his car to admit her invalid chair. For some months he continually declared that he would give his right arm if only his daughter could be cured. Later, while on holiday, his car, with wife and daughter on board, was involved in a crash. The father had his right arm severed at the shoulder. Before the father's wound was healed the daughter's psoriasis and arthritis had disappeared. Dr. Smellie asked whether Dr. MacKenna attributed this to psychological agencies or to liberation of cortisone following adrenal shock.

**Caernarvonshire and Anglesey Paediatric Club.**—The club was formed in December, 1950, to encourage discussion and further knowledge on infant care and management among both lay and medical men. Meetings are held usually at the County Hospital, Bangor, on the third Tuesday in every month at 4.30 p.m., except in June, July, and August. Membership is open to doctors, health visitors, district nurses, and all persons interested in paediatrics, and further information may be obtained from the secretary, Dr. G. W. Roberts, St. Fillans, Segontium Road South, Caernarvon.

**British Rehabilitation Methods Applied in Austria.**—Dr. Walter Ehalt, who is director and chief surgeon of the Graz Accident Hospital, was one of two Austrian medical men to attend the 1948 British Council course on the rehabilitation and resettlement of the disabled. On their return they arranged for the establishment of the first rehabilitation and vocational training centre in Austria on the British model. It was set up by the Austrian Insurance Board, which administers the industrial disability compensation scheme. Dr. Ehalt is now (February 25–March 11) making a further study of British treatment of the disabled at the Royal National Orthopaedic Hospital, the London Hospital, the Wingfield-Morris Hospital, Oxford, the Ministry of Pensions Hospital, Stoke Mandeville, the Birmingham Accident Hospital and Rehabilitation Centre (March 7 and 8), and at Manchester Royal Infirmary (March 10).

**Hospitals Menace Homes.**—The 26 teaching hospitals in London will eventually require another 195 acres of land for their planned extension, which will mean pulling down the houses of 13,800 people to yield the necessary space. The Middlesex Hospital, in Mortimer Street, W.1, is typical. Central London is very densely populated, and planned expansion of the hospital into adjoining property will mean moving 710 people out of 140 houses, 35 shops, two public houses, and seven "other premises." These are some of the startling facts shown by the exhibition of plans, drawings, photographs, and models now at County Hall to illustrate the London County Council's development proposals under the Town and Country Planning Act, 1947. Objections to the plan, which is subject to a quinquennial review, have to be lodged with the L.C.C. by March 16. It is intended to keep the Harley Street district as a medical area.

**Oxford and Cambridge Boat Race.**—Messrs. Vitamins Limited announce that they have available a limited number of tickets for doctors who may wish to view the Boat Race on March 29 from their premises. Early application should be made to Dept. B.R.2, Vitamins Limited, Upper Mall, London, W.6.

**Publications Received.**—*National Register of Medical Auxiliary Services: Speech Therapists:* Board of Registration of Medical Auxiliaries, B.M.A. House, Tavistock Square, London, W.C.1, 1951 (free on application). *Catalogue of Oriental Manuscripts in the Library of the Royal College of Physicians:* reprinted from the *Journal of the Royal Asiatic Society* (apply to Harveian Librarian).

**Emergency Bed Service: Applications and Admissions.**—During the seven days ending March 3 the number of applications made by doctors to the London Emergency Bed Service for admission of patients was 1,138, of whom 87.52% were admitted.

**Will.**—Dr. Frank Hubert Robbins left £30,517. He left £200 to the Royal Medical Benevolent Fund in memory of his son Tony.

## COMING EVENTS

**Postgraduate Course in Venereology.**—The Institute of Urology announces that a postgraduate course on venereology will be held at St. Paul's Hospital, Endell Street, W.C.2, at 11 a.m., from March 3 to 30. Full details from the secretary of the Institute, Henrietta Street, W.C.2.

**Public Health Course.**—The Royal Institute of Public Health and Hygiene announces that the next biannual course of instruction for the Certificate in Public Health, which leads to courses for both the Diploma in Public Health and the Diploma in Industrial Health, will commence on Friday, March 21. All courses may be taken either whole-time or part-time. Prospectuses, enrolment

forms, and full details may be obtained from the secretary of the Royal Institute of Public Health and Hygiene, 28, Portland Place, London, W.1.

**Medical Teaching.**—A conference-course on the technique of medical teaching will be held at the London School of Hygiene and Tropical Medicine, Keppel Street, W.C.1, on March 28 and 29. The programme includes "Teaching Teachers," by Sir Geoffrey Jefferson; "The Influence of Examinations on Teaching," by Professor Harold Rodgers; "The Clinical-pathological Conference," by Professor J. H. Dible; "The Training of the Research Worker," by Professor E. J. Wayne; "Visual Methods," by Dr. R. G. W. Ollerenshaw; "Practical Clinical Instruction," by Professor C. Bruce Perry; and "The Education of the General Practitioner," by Dr. G. O. Barber. Admission by ticket only on application from the Postgraduate Medical School, Du Cane Road, W.12.

**18th International Neurological Reunion.**—This will be held in Paris on July 8 and 9, and will deal with the study of pathological movement. Those interested are requested to register before May 1, and all details may be obtained from M. Jean Sigwald, 68, Boulevard de Courcelles, Paris, 17e.

## SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

### Monday

INSTITUTE OF NEUROLOGY, National Hospital, Queen Square, London, W.C.—March 10, 5 p.m., "*The Physiological Responses of Chromatolysed Neurones*," Guest Lecture by Professor J. C. Eccles, F.R.S.

INSTITUTE OF PSYCHIATRY, Maudsley Hospital, Denmark Hill, London, S.E.—March 10, 5.30 p.m., lecture-demonstration for postgraduates by Dr. E. Stengel.

MEDICAL SOCIETY OF LONDON, 11, Chandos Street, Cavendish Square, W.—March 10, 8.30 p.m., "*Therapeutic Trauma of the Brain*," discussion to be introduced by Dr. G. W. B. James and Dr. P. M. Tow.

### Tuesday

CHELSEA CLINICAL SOCIETY.—At South Kensington Hotel, 41, Queensgate Terrace, London, S.W., March 11, 8.30 p.m., 6th ordinary meeting of 55th session. "*The School Doctor looks Forwards and Backwards*," discussion to be opened by Dr. R. E. Smith.

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—March 11, 5.30 p.m., "*Contact Allergy*," by Dr. S. C. Gold.

MANCHESTER MEDICAL SOCIETY.—At Physiology Theatre, Medical School, Manchester University, March 11, 4.30 p.m., joint meeting of Section of Surgery and Medicine. "*The Treatment of Haematemesis*," by Dr. F. Avery Jones.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, London, S.W.—March 11, 5 p.m., "*The Dynamics of Heart Failure*," Oliver-Sharpey Lecture by Dr. J. McMichael. (See also March 13.)

WEST END HOSPITAL FOR NERVOUS DISEASES, 40, Marylebone Lane, London, W.—March 11, 5.30 p.m., neurological demonstration by Dr. T. Rowland Hill.

### Wednesday

CAMBRIDGE UNIVERSITY.—At Department of Physical Chemistry, Free School Lane, Cambridge, March 12, 12 noon, "*Some Aspects of the Physical Chemistry of Amino-acids, Peptides, and Proteins*," by Professor John T. Edsall (Harvard University).

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—March 12, 5.30 p.m., "*The Origin of Epithelial Neoplasms in the Skin*," by Dr. I. W. Whimster.

INSTITUTE OF NEUROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., March 12, 4.30 for 5 p.m., "*Differential Diagnosis of Renal Pain*," by Mr. J. E. Semple.

LONDON COUNTY MEDICAL SOCIETY.—At Paddington Hospital, Harrow Road, W., March 12, 3 p.m., clinical meeting.

MANCHESTER MEDICAL SOCIETY: SECTION OF PATHOLOGY.—At Medical School, Manchester University, March 12, 4.30 p.m., "*Porphyrins in Health and Disease*," by Dr. J. E. Kench.

### Thursday

ALFRED ADLER MEDICAL SOCIETY.—At 11, Chandos Street, London, W., March 13, 8 p.m., "*The Relationship of Body and Mind*," by Dr. E. K. Ledermann. Discussion to be opened by Dr. E. B. Strauss.

●INSTITUTE OF CHILD HEALTH, Hospital for Sick Children, Great Ormond Street, London, W.C.—March 13, 5 p.m., "*The Ear, Nose, and Throat*," by Mr. H. S. Sharp.

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—March 13, 5.30 p.m., "*Histopathology of Foreign Body and Specific Granulomas*," by Dr. H. Haber.

LIVERPOOL MEDICAL INSTITUTION, 114, Mount Pleasant, Liverpool.—March 13, 8 p.m., pathological meeting.

LONDON JEWISH HOSPITAL MEDICAL SOCIETY.—At Dorchester Hotel, Park Lane, London, W., March 13, 6.30 p.m. to 1 a.m., annual dinner dance.

MANCHESTER MEDICAL SOCIETY: SECTION OF ANAESTHETICS.—At Staff House, Manchester University, March 13, 8.15 p.m., "*The Control of Bleeding During Surgical Operations*," by Dr. W. B. Bacon and Dr. C. E. Sykes.

MEDICAL SOCIETY FOR THE CARE OF THE ELDERLY.—At Old Library, B.M.A. House, Tavistock Square, London, W.C., March 13, 11.30 a.m., Conference on Co-ordinated Medical Care of the Elderly. Introductory Address by Lord Amulree. "*Co-ordinated Medical Care of the Elderly*," to be opened by Dr. J. A. Scott, Dr. E. B. Brooke, and Dr. Barbara Morton. A general discussion will follow. 4.30 p.m., annual general meeting.

ROYAL ARMY MEDICAL COLLEGE.—At Lecture Theatre, John Islip Street, London, S.W., March 13, 5 p.m., "*Common Difficulties in the Diagnosis of Heart Disease*," by Dr. D. Evan Bedford.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, London, S.W.—March 13, 5 p.m., "*The Dynamics of Heart Failure*," Oliver-Sharpey Lecture by Dr. J. McMichael. (See also March 11.)

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—March 13, 5 p.m., "*Ureteric Calculus*," Hunterian Lecture by Professor V. W. Dix.

ST. ANDREWS UNIVERSITY.—At Lecture Theatre, Materia Medica Department, Medical School, Small's Wynd, Dundee, March 13, 5 p.m., "*Transmission of Effects from Nerve Endings—A Survey of the Significance of Present Knowledge*," by Sir Henry Dale, O.M., F.R.S.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL, Hyde Park Corner, London, S.W.—March 13, 4.30 p.m., lecture-demonstration in psychiatry by Dr. D. Curran.

UNIVERSITY COLLEGE, Physiology Theatre, Gower Street, London, W.C.—March 13, 4.45 p.m., "*Mitochondria and Biological Oxidations and Phosphorylations*," by Dr. A. L. Lehninger (Chicago).

UNIVERSITY COLLEGE HOSPITAL MEDICAL SCHOOL, University Street, Gower Street, London, W.C.—March 13, 5 p.m., "*The Structural and Functional Plasticity of Influenza Viruses*," Sydney Ringer Memorial Lecture by Professor Wilson Smith, F.R.S.

### Friday

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—March 14, 5.30 p.m., "*Contact Eczema*," clinical demonstration by Dr. S. C. Gold.

●INSTITUTE OF DISEASES OF THE CHEST AND INSTITUTE OF CARDIOLOGY.—At London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., March 14, 5.30 p.m., "*Surgical Treatment of Mitral Stenosis*," by Mr. R. C. Brock.

OXFORD UNIVERSITY.—At Magdalen College, Oxford, March 14, 5 p.m., "*The Neurophysiological Basis of Mind*," Waynflete Lecture by Professor J. C. Eccles, F.R.S.

## APPOINTMENTS

BUCHANAN, JAMES ANDREW, M.B., Ch.B., D.P.H., County Medical Officer of Health, Banffshire.

EAST ANGLIAN REGIONAL HOSPITAL BOARD.—*Consultant Pathologist to Hospitals in Peterborough and King's Lynn Area*, A. M. Pugh, M.B., Ch.B., D.T.M.&H. *Consultant Radiologist in East Suffolk and Ipswich Area*, K. E. Barlow, M.R.C.S., L.R.C.P., D.M.R., F.F.R. *Consultant Orthopaedic Surgeon in East Suffolk and Ipswich Area*, T. Denness, M.Ch.Orth., F.R.C.S. *Registrar in Psychiatry, Fulbourn Mental Hospital, Fulbourn, Cambs.* Elisabeth Shoenberg, M.R.C.S., L.R.C.P.

FLEMING, G. M., M.D., D.P.H., Medical Officer of Health and School Medical Officer, County Borough of West Bromwich.

GROSS, DESIREE, M.B., M.D., D.P.H., Deputy Medical Officer of Health, Borough of Ilford, and Assistant County Medical Officer, Essex County Council.

LIVERPOOL REGIONAL HOSPITAL BOARD.—*Whole-time Assistant Pathologist to Hospitals in Ormskirk Area*, R. G. Tasker, M.B., B.Chir. *Whole-time Assistant Pathologist to Walton Hospital*, M. J. Wodzinski, M.D. *Whole-time Assistant Psychiatrist to Rainhill Hospital*, G. C. Heller, M.D. *Whole-time Assistant Psychiatrist to Winwick Hospital*, P. M. O'Flanagan, L.R.C.P.&S.I., D.P.M. *Whole-time Consultant Psychiatrist to Rainhill Hospital*, S. Falk, M.D., D.P.M. *Consultant Surgeon to Regional Thoracic Unit*, J. K. B. Waddington, F.R.C.S.Ed.

MCCOLL, MICHAEL, M. B., Ch.B., D.P.M., Deputy Medical Superintendent, and Consultant Psychiatrist, Barnsley Hall Hospital, Bromsgrove, Birmingham Regional Hospital Board.

MANCHESTER REGIONAL HOSPITAL BOARD.—*Consultant Psychiatrist, Preston and Chorley Hospital Centre, and Whittingham Hospital*, C. S. Parker, M.D., D.P.M. *Consultant General Physician, Macclesfield Hospital Centre*, F. W. Smith, M.B., Ch.B., M.R.C.P. *Consultant Obstetrician/Gynaecologist, Crewe Hospital Centre*, H. D. Freeth, M.D., M.R.C.O.G. *Consultant General Physician, Crumpsall Hospital, Manchester*, M. H. Oelbaum, M.D., M.R.C.P. *Consultant Obstetrician/Gynaecologist, Park Hospital, Davyhulme*, D. C. A. Bevis, M.B., Ch.B., M.R.C.O.G. *Assistant Anaesthetist, Rochdale Hospitals (S.H.M.O.)*, S. Mehlmann, M.D. *Assistant Psychiatrist and Deputy Medical Superintendent, Calderstones Hospital (S.H.M.O.)*, E. M. Hutton, M.B., B.Ch. *Tuberculosis Physician, Wigan and Leigh Hospital Areas*, C. D. Rushworth, M.R.C.S., L.R.C.P.

SHIEL, JOHN B., M.B., Ch.B., D.P.H., County Medical Officer, Stewartry of Kirkcudbright.

SOUTH-WEST METROPOLITAN REGIONAL HOSPITAL BOARD.—*Whole-time Deputy Physician-Superintendent and Consultant Psychiatrist, St. Lawrence's Hospital, Caterham,* J. J. Ryan, M.B., B.Ch., D.P.H., D.P.M. *Part-time Assistant Ophthalmologists (S.H.M.O.), Portsmouth Group of Hospitals,* T. G. S. Murray, M.R.C.S., L.R.C.P., D.O.M.S., A. E. Barrett, M.R.C.S., L.R.C.P., D.O.M.S. *Whole-time Assistant Radiotherapist (S.H.M.O.), Lambeth Hospital,* R. Parfitt, M.R.C.S., L.R.C.P., D.M.R.T. *Part-time Consultant Orthopaedic Surgeon, St. Helier Group of Hospitals,* H. H. Kennedy, F.R.C.S.Ed. *Part-time Consultant Surgeon, Portsmouth Group of Hospitals,* W. W. Wiggins-Davies, M.Chir., F.R.C.S. *Part-time Consultant Dermatologist, Portsmouth and Isle of Wight Groups of Hospitals,* A. A. C. Ross, M.B., Ch.B., M.R.C.P.Ed. *Registrar to Medical Department, Royal Victoria and Christchurch Hospitals, Bournemouth,* C. C. Downie, M.B., Ch.B. *Registrar to Surgical Department, St. Luke's Hospital, Guildford,* J. F. Shaw, M.B., B.S. *Senior Registrar to Orthopaedic Surgery Department, Southampton and Isle of Wight Groups of Hospitals,* E. H. J. Smyth, M.B., B.S., F.A.C.S. *Registrar to Surgical Department, St. Stephen's Hospital,* J. L. Swann, M.B., B.S., F.R.C.S. *Registrar to Obstetric and Gynaecological Department, Redhill County Hospital,* M. G. Pearson, M.B., Ch.B., M.R.C.O.G. *Registrar to Pathological Department, Royal Victoria Hospital, Bournemouth,* Eilean A. Hunter, M.B., Ch.B. *Registrar to Orthopaedic Surgery Department, Lord Mayor Treloar Orthopaedic Hospital, Alton,* I. G. MacKenzie, M.D., F.R.C.S.Ed. *Senior Registrar to Thoracic Surgery Department, Southampton Infectious Diseases Hospital,* G. Katz, M.B., B.Ch., F.R.C.S. *Registrar to Anaesthetic Department, Queen Mary's Hospital, Carshalton,* M. Flynn, M.B., B.Ch.

TRYTHALL, D. A. H., M.D., M.R.C.P., D.C.H., Assistant Medical Registrar, Hospital for Sick Children, Great Ormond Street, London, W.C.

WILSON-SHARP, CECIL DEREK, M.B., B.S., D.M.R.D., Senior Registrar, Department of Radiodiagnosis, The London Hospital, E.

## BIRTHS, MARRIAGES, AND DEATHS

### BIRTHS

**Alexander.**—On February 22, 1952, to Nancy (formerly Walmsley), wife of Francis G. Alexander, M.B., Ch.B., of Small Heath, Birmingham, a sister for David and Alison.

**Orford Smith.**—On February 15, 1952, at the Queen Elizabeth Hospital, to Mary, wife of Dr. E. S. Orford Smith, of 59, Hagley Road West, Harborne, Birmingham, a sister for John David and Margaret.

### DEATHS

**Ainsworth.**—On February 13, 1952, at the Royal West Sussex Hospital, Hugh Ainsworth, M.B., F.R.C.S., Colonel, I.M.S., retired.

**Armstrong.**—On February 19, 1952, at Braintree, Essex, John Thomas Armstrong, L.R.C.P.&S.Ed., L.R.F.P.S., of the Constitutional Club, London, W.C.

**Brown.**—On February 16, 1952, William Brown, L.R.C.P.&S.Ed., L.R.F.P.S., of Burbage Road, London, S.E.

**Campbell.**—On February 16, 1952, John Alexander Langford Campbell, O.B.E., M.D., Surgeon Captain, R.N., retired, of 24, Norfolk Road, London, N.W.

**Cuthbert.**—On February 20, 1952, at Edinburgh, James Mackie Cuthbert, M.B., Ch.B., Major, R.A.M.C., retired.

**Davies.**—On February 20, 1952, at Moranedd, Towyn, Merioneth, John Alban Davies, L.M.S.S.A.

**Devlin.**—On February 17, 1952, at Old Chapel House, Richmond Hill, Leeds, James Francis Patrick Devlin, M.B., B.Ch., aged 59.

**Douglas.**—On February 2, 1952, at Hamilton, New Zealand, Mary Douglas, M.B., Ch.B., D.R.C.O.G.

**Dowler.**—On February 19, 1952, at The Limes Nursing Home, Burgess Hill, Sussex, Herbert Matthew Dowler, M.R.C.S., L.R.C.P., of Mussoorie, United Provinces, India, aged 83.

**Eames.**—On February 15, 1952, at Shrewsbury, John Ward Eames, M.B., Ch.B., Colonel, A.M.S.

**Graves.**—On February 11, 1952, at Worthing, Sussex, Charles Frederick Graves, L.R.C.P.&S.Ed., L.R.F.P.S., formerly of Paddington, London, W.

**Hall.**—On February 15, 1952, at 9, Delfield Close, Watford, Herts, Charles Herbert Hall, M.D., aged 84.

**Hartley.**—On February 18, 1952, at 29, Lower Oxford Street, Castleford, Yorks, Walter Dixon Hartley, M.B., B.S., aged 74.

**Hicks.**—On February 12, 1952, Charles Edward Hicks, M.R.C.S., L.R.C.P., of St. Ives, Huntingdon, aged 77.

**Hobbs.**—On February 21, 1952, at Trundle Nursing Home, Caterham, John Hobbs, L.R.C.P.&S.I. & L.M., Major R.A.M.C., retired, of 22, Timber Hill, Caterham, Surrey, aged 89.

**Leckie.**—On February 18, 1952, at Perth, Joseph Primrose Leckie, M.B., F.R.C.P.Ed., of the Hydropathic, Crieff, Perthshire.

**McCalden.**—On February 13, 1952, Joseph Crawford Alfred McCalden, M.B., B.Ch., of 60, Kingston Crescent, Portsmouth.

**Matheson.**—On February 3, 1952, Ian Henry Matheson, M.B., Ch.B., of Coombe House, Halberton, Tiverton, Devon, aged 44.

**Morcom.**—On February 12, 1952, at 41, South Eaton Place, London, S.W., Alfred Farr Morcom, M.B., B.Ch.

**Nash.**—On February 8, 1952, at Strawberry Cottage, Sandhurst, Camberley, Berks, Charles Henry Nash, L.R.C.P.&S.Ed., L.R.F.P.S.

**Orr.**—On February 15, 1952, at Edinburgh, William Basil Orr, M.D., of 2, South Learmonth Avenue, Edinburgh.

**Patton.**—On February 20, 1952, William Patton, M.B., Ch.B., of Dunarnon, Amersham, Bucks.

**Pearson.**—On February 18, 1952, at Greystead, Blackwell Scar, Darlington, Henry Carden Pearson, M.B., F.R.C.S.Ed., aged 77.

**Scott.**—On February 4, 1952, at Ruan High Lanes, Truro, Cornwall, Frank Sholl Scott, M.B., B.S., aged 68.

**Spence.**—On February 7, 1952, at Bognor Regis, Sussex, Harold Douglas Livingstone Spence, M.D., M.Ch.

**Thomas.**—On February 6, 1952, at the home of his nephew, 15, Prospect Row, Brompton, Chatham, Kent, John Henry Thomas, M.R.C.S., Fleet Surgeon, R.N., retired, aged 94.

**Tripp.**—On February 2, 1952, Charles Llewellyn Howard Tripp, M.R.C.S., L.R.C.P., late of the Falcon Hotel, Bude, Cornwall, aged 94.

**Waterfield.**—On February 6, 1952, at Beach Haven, Hooe, Plymstock, Devon, William Henry Waterfield, L.R.C.P.&S.I. & L.M., late of Stonehouse, Plymouth, aged 89.

**Wells.**—On February 17, 1952, at Tylers Cross, Roydon, Essex, John Edmund Bishop Wells, M.R.C.S., L.R.C.P., Major, R.A.M.C., T.F., retired.

**Willis.**—On February 10, 1952, at 96, Evington Drive, Leicester, David Victor Stewart Willis, M.B., B.Ch., late of Walsall, Staffs, aged 65.

# Any Questions?

*Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.*

### Night Nurses' Paralysis

**Q.**—*What is the explanation of night nurses' paralysis?*

**A.**—Night nurses' paralysis is a frightening but banal condition. It usually occurs during the early hours of the morning when the nurse is sitting at her table in the darkened ward; suddenly someone enters the ward or a patient shouts for assistance and the nurse finds herself quite unable to move or even to speak for some seconds, though the period of "paralysis" may seem to be much longer than this. Suddenly power returns and she is able to carry on with her duties. This condition is the same as the "sleep paralysis," which is not uncommon—that inability to move, often accompanied by anxiety, which may occur as a person is just going off to sleep or more commonly on first awakening. The explanation seems to be that the motor and postural centres "fall asleep" before the mind, or during the process of waking the mind "wakens" before the motor and postural centres (*British Medical Journal*, 1939, **2**, 51). The problem is of course more involved than this: there is obviously similarity to cataplexy with the transient loss of power and tone in response to a sudden emotional stimulus, and it may also be significant that attacks of familial periodic paralysis which are recognized to be associated with a fall in the blood potassium are nearly always at their maximum intensity on first waking in the morning. More knowledge of the actual physiology of sleep is necessary before a complete answer can be given to this question.

### Psychological Effects of Female Castration

**Q.**—*How real is the risk that a bilateral oophorectomy will have serious psychological consequences in a woman of 41?*

**A.**—Bilateral oophorectomy carried out in a woman aged 41 years will almost certainly be followed by some menopausal symptoms. These will mainly take the form of hot flushes, and their severity and duration are variable. Symptoms are severe in about half the cases. In a stable woman there is no need to fear serious nervous reaction, possibly some temporary emotional upset at most. In an unstable woman, however, the risk is much higher and depends on the degree and type of the present instability. If she has been subject to mental disorder in the past the operation might well cause a relapse. Each case needs to be judged on its merits, and the outlook of individual patients on the menopause is important.

### Pneumonectomy for Bronchiectasis

**Q.**—*What are the indications for and against lung resection in the treatment of bronchiectasis?*

**A.**—Two factors play important parts in the selection of cases for lung resection: (1) The distribution and extent of the bronchiectasis: the more localized and limited the disease the better for surgery. (2) The age of the patient and the ability to survive on what lung tissue is left behind. The ideal case is that of a child with cough and sputum and with the disease limited to a lobe. Here the results of surgery are excellent. Elderly, emphysematous patients with diffuse bilateral disease are not suitable.

In assessing a patient complete bilateral bronchograms are necessary, and gross upper respiratory infection must be eliminated or reduced. Also pulmonary tuberculosis should be excluded. Patients with toxaemia and excessive sputum are in more urgent need of surgery than those whose symptoms are less obvious. Bronchiectatic changes in both lungs are less favourable than when the same amount of disease is concentrated in one lung, but limited bilateral operations in patients under middle age are quite practicable. As much as one lung or three lobes (the lingula being counted as a lobe) may be removed, particularly if the disease is gross and the lung has been rendered functionless. If the disease is diffuse and involves only parts of lobes, several segmental resections can be performed, but diffuse disease is more likely to be progressive than lobar bronchiectasis, which may have had its origin in bronchial obstruction by enlarged hilar glands. Too much attention need not be paid to the type of bronchiectasis—e.g., saccular, cystic, or fusiform; it is the symptoms and extent of the change that count. An age limit of about 40 years is usual, though in the absence of emphysema and with supple chest movements this may be extended. At the other extreme, if possible, operation should be postponed until a child can co-operate in the postural and physical treatment that are essential both before and after operation.

### Is Bread Good for Diabetics?

**Q.**—*Is bread a good food for diabetics? If not, what alternatives are suggested?*

**A.**—Bread is an excellent food for a diabetic patient to eat. One ounce (28.4 g.) of our present national wheatmeal (85% extraction) contains 15.7 g. of carbohydrate, expressed as a glucose, and about 2 g. of protein; a white bread (70% extraction) contains rather more carbohydrate—16.8 g. per ounce; and "hovis" bread less, at 13.35 g. per ounce. Patients do not often change the kind of bread they eat, and in any case the small difference in carbohydrate content of different breads does not matter when the diet contains, as it should do, 150–200 g. or more of carbohydrate. If, however, a patient who, say, eats only hovis bread travels abroad and eats the same amount of white bread exclusively, the urine might contain a little sugar, which would necessitate 2 or 4 units more insulin. On returning to the hovis bread slight symptoms of hypoglycaemia might occur unless the dose of insulin was promptly reduced.

### Treatment of Congenital Absence of Rectum

**Q.**—*What recent advances have there been in the treatment of congenital absence of the rectum? Is there any satisfactory alternative to a permanent colostomy?*

**A.**—Whereas it was formerly believed that there was no satisfactory alternative to colostomy in cases of imperforate anus with absence of the rectum, it does now seem that restoration of continuity with a properly functioning anus is possible in some cases. Clearly such restorative operations are only possible where the sphincteric apparatus has developed normally. Under these conditions there are some authorities[1] who state that repair can be effected by a purely perineal approach, even without a preliminary colostomy. The rectal pouch has to be freely mobilized so that it can be brought down to the anus without tension; from the technical point of view the operation can be very difficult and may be succeeded by stricture at the suture line. At the best, a functioning anus will be attained; at the worst, the patient may be left with a perineal colostomy, possibly associated with stricture formation. To facilitate repair in those cases where the rectum ends blindly at the pelvic floor a synchronous combined abdomino-perineal procedure has been described.[2] It must be emphasized, however, that expert opinion in this country is inclined to the view that, desirable though they may be, these operations are often not feasible. In general, restorative operations should be undertaken as soon after birth as possible, and it is most unlikely that the sphincteric apparatus would function.

REFERENCES

1 Wallace, F. T., and Calvin, E. M. (1948). *Surgery*, 24, 832.
2 Rhoads, J. E., Pipes, R. L., and Randall, J. P. (1948). *Ann. Surg.*, 127, 552.

### Physical Characters Associated with Schizophrenia

**Q.**—*With what physical characteristics is a tendency to schizophrenic illness genetically associated? Brown eyes, for instance, appear to be rare in schizophrenics.*

**A.**—So far as is known there is no single physical trait genetically associated with schizophrenia. There is evidence that tuberculosis is abnormally frequent, not only among schizophrenics themselves, but also among their brothers and sisters; but parallel investigations have failed to show any increased tendency to schizophrenia among the tuberculous or their relatives. It may be that schizophrenics in this country are rather rarely brown-eyed; but it must be remembered that schizophrenia is no less frequent in countries such as Greece and Italy, where the bulk of the population is brown-eyed, than it is here.

### Television and the Eyes

**Q.**—*Is television dangerous to the eyes?*

**A.**—No. If any significant refractive error is present, correcting spectacles should obviously be worn.

## NOTES AND COMMENTS

**Pills for Bankrupt Smokers.**—In reply to a question about the dosage and frequency of administration of the pills for preventing the desire to smoke ("Any Questions?" January 12, p. 118), OUR EXPERT suggests that 5-gr. (0.32-g.) pills might be taken three or four times a day for as long as required.

**Multiple Myelomatosis.**—Dr. A. PINEY (London) writes: The answer to the question on multiple myelomatosis ("Any Questions?" February 9, p. 340) is a little misleading in its description of the diet during treatment with stilbamidine. The paper in which this was advocated came from the United States, and the diet suggested was one which for that opulent country was low in protein but for us is at about the level of our rations. There is thus no reason why patients with myelomatosis should have their protein intake reduced below British standards. Further, the answer does not mention that preliminary treatment with stilbamidine greatly increases the radiosensitivity of the neoplastic cells.

**Angina in Cold Weather.**—Dr. G. BILSBARROW (Ontario, Canada), commenting on a reply to a question asking whether it would be advisable for a lady suffering from angina pectoris, aggravated by cold, to emigrate to Ontario ("Any Questions?" January 12, p. 118), writes: Having been in practice in Northwest Ontario for the past three years, where a temperature range of 90° F. (32.2° C.) in summer to 40° F. below zero (−40° C.) in winter is common, I have not noted any increase in symptoms of angina among our patients. Most of the work here is outdoors and we have a normal incidence of ischaemic heart disease, but this is neither increased in winter nor is there any increase in the incidence of symptoms among known cases.

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 4499. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.
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